Exploring Relationships between Stress, Sleep and Body Weight in Teenagers: A Longitudinal Study

By Marthe Fjellberg Magerøy and Veronica Holloway

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Abstract

Excess weight gain in children and adolescents are steadily increasing throughout the world and brings about adverse health effects. Recent findings suggest that stress might be a promoting factor for the development of excessive weight gain. Adolescents may be especially vulnerable and sensitive to stress exposure. In addition, parallel to the rise of obesity, there has been a related growth in shortened sleep duration and sleep may be an independent risk factor in the development of weight gain, but may also be seen as an indication of stress. However, little is known about the effects of stress and sleep in relation to weight gain, particularly with respect to adolescents and over time.

Thus, the present study aims at exploring the influence of various stress predictors on body weight among adolescents in Oslo, over time. More specifically, investigate the influence of "Life Stress", "Daily Hassles", "Violence", and "Sleep Duration", in relation to body weight, expressed by BMI status, over three different test times (2006, 2008, and 2009).

The population consisted of approximately 2000 high school students from Oslo. The study design is longitudinal with a quantitative approach.

Multiple stepwise backward linear and logistic regression analysis found that Life Stress predicted higher BMI in girls, and not boys, three years later. Decreased Sleep Duration predicted higher BMI in boys, and not in girls.

These stress related gender differences needs to be considered in preventative efforts entailing weight reduction.

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Are teenagers becoming overweight due to stress and inadequate sleep? In the past 30 years, there has been an alarming increase in the numbers of people in populations of developed nations who are either overweight or obese. Over nutrition and obesity have been transformed from relatively minor public health issue, which primarily affects the most affluent societies, to a major public health threat that are being increasingly seen throughout the world. According to the Worlds Health Organization (WHO) overweight and obesity are now a global health problem. They have estimated that more than 1.5 billion adults are overweight and close to 500 million of them are obese – worldwide (The World Health Organization, 2011b). The problem is not only related to adults, on the contrary, obesity in children is already considered an epidemic in many parts of the Western world, and increasing steadily in others. They further estimated that close to 43 million children, less than 5 years of age were considered overweight in 2010.

Recently, increased attention has been given to the potential role of stress as a promoting factor for the development of excessive weight gain. There are multiple types of studies, across different scientific disciplines, which reports evidence; linking stress to weight gain and obesity (De Vriendt, Moreno, & De Henauw, 2009; Torres & Nowson, 2007). Certain age groups also appear to tolerate or cope with stress differently, and newer evidence actually suggests that adolescents' may be particularly vulnerable and sensitive to stress exposure (Lupien, McEwen, Gunnar, & Heim, 2009; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010). In fact, Lupien et al., (2009) found evidence that stress levels may be twice as high in adolescents, than in adults with similar stress experiences, and the effects appears to be long lasting. Another relevant phenomenon is sleep or lack there off. Interestingly, parallel to the rise of obesity, there has been an associated growth in shortened sleep duration. Several systematic reviews suggest that inadequate sleep may be an independent risk factor in the development of weight gain and obesity (Cappuccio et al., 2008; Carter, Taylor, Williams, & Taylor, 2011; Patel & Hu, 2008). Inherently, stress and sleep influences needs awareness in terms of weight related research.

The Weight Phenomena & Related Consequences

The growing science on this matter confirms that being overweight or obese; are related to numerous negative and serious health effects within multiple domains. As well as robust data linking increased body weight, with increased morbidity, and higher mortality rates (Lewis et al., 2009). Obesity related co-morbidities are now starting to surface in childhood, whereas before; it

was mainly related to adults. Due to the novelty of this phenomenon, evidence based data on immediate and long-term health consequences are lacking and will become an additional challenge in the near future (Must, Jacques, Dallal, Bajema, & Dietz, 1992). Yet, newer research highlights that children and adolescents are more vulnerable and sensitive to weight influences, they have a higher likelihood of weight problems as adults, and are more likely to suffer from physical, psychological, and social impacts – related to excessive weight (Baird et al., 2005; Puhl & Latner, 2007).

From a societal and political viewpoint, the obesity related health care costs are substantial. For example, results from a recent review in Europe, Müller-Riemenschneider, Reinhold, Berghöfer, and Willich (2008) found that about 10.4 million euros were expended on obesity-associated healthcare. Extensive costs associated with obesity are found in the United States as well, and a recent study documented that close to 1.2 percent of gross domestic product were spent on obesity related healthcare (Yach, Stuckler, & Brownell, 2006).

The complications of excessive weight during all stages in life can mainly be classified as physical and psychosocial.

Physical Consequences

An exhaustive body of literature points out that being overweight and/or obese are related to many serious health consequences, such as; cardiovascular diseases; diabetes; musculoskeletal disorders; and some types of cancers. In addition, there may also be a higher risk of premature death; problem with breathing; increased risk of fractures; high blood pressure; and disability in adulthood (Puhl & Latner, 2007; The World Health Organization, 2011a; Wilfley, Vannucci, & White, 2010).

Psychosocial Consequences

There are also substantial reasons to be worried about the immediate and long-term effects of elevated BMI with respect to well-being and quality of life. Puhl and Heuer (2009), claims that weight bias or stigma is often related to negative stereotypical viewpoints such as; being less competent; lazy; and so on. These negative attributes are thought to be within the person's control and as a consequence; overweight/obese people are judged and treated more harsh by the public.

Defining Overweight and Prevalence

'Overweight' refers to weighing more than a standard level for height and age and World Health Organization (2012) defines overweight and obesity on their website as: "…*abnormal or excessive accumulation of fat that may impair an individual's overall health***".** There are also multiple anthropometric indices used for defining weight status and predicting health related risks associated with overweight and obesity status. The Body Mass Index, hereafter referred to as BMI, is a useful tool and classification system which is often utilized to show and predict the balance between height and weight, and it is often used in population based studies (Sebo, Beer-Borst, Haller, & Bovier, 2008; The World Health Organization, 2011a). The cutoffs in this classification system are set based on co-morbidities risks that are associated with BMI (Guh et al., 2009). Although international growth standards utilizing the BMI on school-age children and adolescents have identified possible weaknesses with this index (Butte, Garza, & de Onis, 2007). The most common known BMI weakness relates to the fact that it does not differentiate between muscles and fat.

Systematic weight measurement studies on Norwegian children and youth, is currently lacking. Given the public health concern associated with excessive weight, the Norwegian - Directorate of Health, has recently implemented new systematic weight measurement guidelines among Norwegian children. Children and adolescents' growth status will now be monitored on a regular basis at health clinics and in school-health services. Nevertheless, the few and recent studies available on Norwegian youth populations does confirm these negative weight trends. Population studies from Statistics Norway, recently reported that 25 percent of those over 16 years of age were overweight (Wilhelmsen, 2009). According to Hovengen (2011), 19 percent of the girls and 12 percent of the boys in third grade were defined as overweight and 3 percent of the girls and 5 percent of the boys fit the criteria for obese status.

Predictors and Causes of Weight Gain and Obesity

The predictors of elevated adipose tissue are complex and multifaceted. However, the proximal cause is a growing imbalance between energy intake or a downward shift in total energy expenditure, or both. Distal causes of the obesity epidemic include changes in behavioral and environmental factors that may stimulate energy intake or depress energy expenditure. In other words, increased consumption of energy-dense foods high in saturated fats and sugars, and

reduced physical activity (The World Health Organization, 2011a). Despite the alleged dietary and activity risk factors – widely accepted, research which focuses on adolescents over time, have not been able to find strong or consistent support for this (Haines, Neumark-Sztainer, Wall, & Story, 2007; Rehkopf, Laraia, Segal, Braithwaite, & Epel, 2011; Stice, Presnell, Shaw, & Rohde, 2005; The, Suchindran, North, Popkin, & Gordon-Larsen, 2010). In fact, the same researchers paradoxically have found that having weight concerns (e.g., body dissatisfaction and drive for thinness), dieting, and practicing weight control behavior (e.g., laxatives, diuretics, and vomiting) at the first test time 1, strongly relate to obesity at the second test time 2. These tendencies were found amongst both sexes.

An individual's weight is undoubtedly also the result of genetic components. Sadly, offspring of obese parents are consistently at increased risk of being overweight and this is a consistent finding among studies (Parsons, Power, Logan, & Summerbell, 1999; The World Health Organization, 2011b). The heritability of excess body weight has been studied in family, twin and adoption studies. The latter, shows that the genetic effect on weight and height is fully expressed in childhood, and that children have a weight and height closer to their biological parents, than to those that raised them (Sørensen, Holst, & Stunkard, 1992). The debate on genetics versus environment influences is still an active theme in overweight and obesity research. However, despite the often high genetic component widely documented (heritability estimates of 30 – 70 percent), newer research often suggests a Gene - Environment Interaction. This refers to a situation in which the response or the adaptation to an environmental agent, a behavior, or a change in behavior is conditional on the genotype of the individual (Chan, 2010). In other words, genetic factors alone cannot explain the recent explosion in weight gain - globally, however, since the environment and behavior has changed, the genes' influence on obesity has probably also changed.

Depressive symptoms and BMI are often found to be positively associated in studies on adolescents and over time, however, there are inconsistent results in regards to gender tendencies (Goodman & Whitaker, 2002; Haines et al., 2007; The et al., 2010). On the other hand, Luppino and fellow researchers (2010), in a systematic review and meta-analysis on longitudinal relations, validate a bidirectional link between depression and elevated BMI, in both adult women and men. Although the study did not find the same effect in participants below 20 years

of age, they propose that the "time factor" may be involved, and possibly explains the age variation. Thus, time could be thought of as a third variable responsible for the inconsistent gender results in an already irregular puberty phase. Interestingly, it also appears that depression and excessive weight share common determinants, mainly dysregulation of the stress system. A recent study, found that the association between depression and BMI was mediated by a stress hormone (Dockray, Susman, & Dorn, 2009).

Socio-economic status (SES) and elevated BMI has also been proposed as a predictor of obesity, however, Wang (2001) found no consensus of this, and proposes that SES in relation to BMI varies across countries.

Haines and fellow researchers (2007) examined the influence of psychosocial factors within the family unit on weight gain. Their data indicated that pressures from parents to lose weight or weight-related teasing were predictive of becoming overweight. Thus, receiving pressure at home, even though the motivation is meant well, may actually be counterproductive and harmful.

Evidence based data from school-age children and adolescents' are currently sparse. In particular analysis over time, which would be especially useful, as they may help to shed light over some factors which are promoting overweight and obesity.

Stress and the Relation to Unhealthy Weight Gain

Recently, increased attention has been given to the potential role of chronic stress as a promoting factor for the development of overweight and obesity in the obesogenic world environment (Dallman, Pecoraro, & la Fleur, 2005; Tsigos & Chrousos, 2006). Abnormal weight gain may be linked directly to increased energy intake and decreased energy expenditure, both of which may possibly reinforce additional weight gain. And a recent review suggests that stress, more specifically chronic stress, may be interacting with both mechanisms of energy imbalance (De Vriendt et al., 2009). However, the mechanisms underlying the effect of stress, in relation to weight gain, are still not completely understood. Stress in itself, can have negative health consequences that have a wide spread and detrimental effect upon bodily systems (American Psychological Association, 2011).

The American Psychological Association (APA) uses Baum (1990) when defining stress: " *... a negative emotional experience accompanied by predictable biochemical, physiological and*

behavioral changes.." (p. 653). Stress is a general term used for the overall concept that diverse perceived threatening stimuli or stressors influence the stress system in the human body and in this way induces a stress response. Acute stress is a relatively brief state of arousal and generally it has an unambiguous beginning and counteraction patterns. Chronic stress on the other hand, is a continuous state of arousal, and the individual may perceive the demands as beyond the inner and outer resources one has available to cope with them (American Psychological Association, 2011).

Genetic, environmental and developmental factors determine the adaptive response of an individual to a stressor (Charmandari, Kino, Souvatzoglou, & Chrousos, 2003). Stressors can be any phenomenon that triggers the stress system, they may take very diverse forms; they may not only be psychological (e.g. divorce, loss of family, demanding job, etc.) or social (e.g. low social position and lack of friends), they can for instance be of physical nature (e.g. noise and injury), of a chemical nature (e.g. smoke or alcohol) or of a biological nature (e.g. bacterial or high blood pressure). However, psychological and social stress is especially characteristic for humans, and psychological stress is often defined as "psychosocial" since it is generated from the social environment (Pollard, 1997). In the western societies, psychosocial stress is increasing (Wittchen & Jacobi, 2005) also putting more pressure on the life of children and adolescents (Barlow & Underdown, 2005).

From here on, before an exploration of the literature on the possible relationship between stress and body weight, stress explanatory factors and different stress indications, including sleep, will be examined.

A Closer Look upon Stress Explanatory Factors

Stress appears to activate inner physiological responses that are meant to restore homeostasis and promote survival of our species. When one encounters stressors, the stress system translates these into an appropriate stress response by temporarily giving priority to certain functions and physiological systems in the body and temporarily suppressing other. (Charmandari, Tsigos, & Chrousos, 2005). A well-known and accepted physiological explanation to the negative effects of stress is the activation of the two key systems norepinephrine–sympathetic adrenomedullary (NE-SAM) system and particularly - the hypothalamus-pituitary-adrenal (HPA) system. Stress induced stimuli, which disrupt the optimal

functioning of these systems have been identified to play an important role in preferential body fat accumulation. The NE-SAM (SNS – sympathetic nervous system) system produces the neurotransmitters epinephrine and norepinephrine, and these are in turn primarily responsible for the fight or flight response (Cannon, 1929; Sherwood, 2001). Stimulation of the HPA axis by any stressor leads to the production and the release of cortisol (also referred to as glucocorticoids in literature), from the adrenal glands. It is classified as a steroid hormone and has a critical and widespread effect - upon both body and brain. As a matter of fact, when this system is activated it may affect almost every nucleated cell in the body, including emotions and cognitive processes. When activated, the heart rate is elevated to increase energy resources, fat and protein stores are metabolized, digestion goes down and/or stop, immune system responses are inhibited, as well as the growth system (Charmandari et al., 2005; Chrousos, 2000; Gunnar & Cheatham, 2003; Kudielka & Kirschbaum, 2005; Meaney et al., 1996). Any impairment or abnormalities with the HPA system are associated with and can cause numerous psychosomatic diseases and psychiatric disorders (for reviews on hyperactive and hypoactive HPA system and related illnesses see: Kudielka et al., 2005). Charmandari and fellow researchers (2005) have found that the stress axes, SNS and HPA, are tightly interconnected at several levels, and are often activated simultaneously. Despite the fact that the stress system is essential for survival, chronic persistent and/or repeated stressors may lead to an overstimulation of the stress system and this may result in an increased and prolonged secretion of CRH and glucocorticoids (Charmandari et al., 2003; Charmandari et al., 2005). It is important to note however, that cognitive interpretations of the stressor also affect the subsequent physical reaction. Therefore, according Lazarus and Folkman (1984), the physical reaction to a stressor is individual – depending partially on coping style.

Daily Hassles and Life Events

Until the 1980's, one of the most salient features of stress research was its focus on dramatic life events: *"environmental circumstances that have an identifiable onset and ending and may carry the potential for altering an individual's present state of mental and physical well-being (p. 204)"(Goodyer, 2001; A. D. Kanner, Coyne, Schaefer, & Lazarus, 1981a).* In contrast to this major life events approach, Lazarus and colleagues suggested vast adaptational significance of the relatively minor stresses and pleasures that characterize everyday life (Coyne, 1979; A. Kanner & Coyne, 1979; R. S Lazarus, 1980; R. S. Lazarus, Kanner, & Folkman, 1980).

Kanner et al. (1981a) termed these everyday incidences daily "hassles" and "uplifts" and defined daily hassles as *"irritating, frustrating, distressing demands that to some degree characterize everyday transactions with the environment"* (p. 3). They include frustrating practical problems such as loosing something or traffic jams, and unexpected occurrences such as bad weather, as well as disappointments, quarrels and financial and family or partner concerns. Most likely, no person leads a hassle-free life, therefore, the impact of hassles on physical and mental health, if any, should depend on factors such as a repeatedly high frequency of hassles, how intense the hassles are during a given period, or the presence of one or a few repeated hassles of strong psychological importance (Kanner et al., 1981). Hinkle (1974) has proposed that major life events might operate by affecting health through the interference of social relationships, habits, and patterns of activity, in addition to the health related behaviors associated with them. In other terms, a person's pattern of daily hassles could be affected by major life events.

Living in an urban setting might increase number of hassles experienced. A study in Britain, found that urban participants had higher rates on scales measuring mental symptoms of distress, than rural participants (Paykel, Abbott, Jenkins, Brugha, & Meltzer, 2000). Furthermore, Lederbogen et al., (2011) recently found evidence on physiological changes in the brain structures and functions, taken with functional magnetic resonance imaging (fMRI). These changes were linked towards higher levels of social stress amongst the participants in the experimental group - urbanized participants, compared to the control group - rural participants. Adolescents may be particularly vulnerable to this effect. In New Delhi- India, adolescents whom scored higher on urban stress were shown to have more negative health habits (Suchday, Kapur, Ewart, & Friedberg, 2006). In contrast, a national Norwegian study unexpectedly found that young urban participants scored higher on sense of mastery (a measurement on stress) than rural participants did (Clench-Aas, Rognerud, & Dalgard, 2009). Subsequently, whether stressful life-events and/or urban hassles will influence Norwegian school children's BMI, will remain to be seen.

Bullying and Violence

Chronic difficulties such as being bullied or experiencing a violent environment on a regular basis can also be seen as hassles that induce stress responses, and which may increase vulnerability to negative health symptoms (Ewart & Suchday, 2002). Bullying can be defined as

all forms of repeated physical or mental violence performed by an individual on another person who is not capable of defending him/herself (Roland & Idsøe, 2001). Being victimized may generate a great deal of distress in a child, and children involved in bullying (victims, bullies or bully/victims) may exhibit a large number of psychosomatic symptoms and health problems (Houbre, Tarquinio, Thuillier, & Hergott, 2006). WHO (1996) defines violence as "*the intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in, or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation*" (p. 1084). Experiencing violence regularly might be stress provoking and lead to bad health. It is important to note, however, that the correlation between violence and stress is not causal. In contrast to happier peers, somewhat melancholic adolescents might be more alert to violence in the environment (Ewart & Suchday, 2002). According to Ozer and Weinstein (2004), greater exposure to violence are linked to more self-reported symptoms of Post-traumatic Stress Disorder (PTSD), and depression for young adolescents in both boys and girls. Also, several studies have found relationships between hostility and Coronary Heart Disease (Miller, Smith, Turner, Guijarro, & Hallet, 1996a, 1996b). Importantly, childhood exposure to violence has been shown to be a risk factor for developing obesity (Felitti et al., 1998).

Sleep as an Indirect Measure of Stress

Several studies have found connections between sleep and stress: stress is strongly linked to the quality of nighttime sleep and impaired awakening (Akerstedt et al., 2002; Morin, Rodrigue, & Ivers, 2003). Also, subjective reports of sleep disturbance indicate that 70 to 91% of patients with PTSD have difficulty falling or staying asleep (Maher, Rego, & Asnis, 2006). Yet another study found that mindfulness-based stress reduction significantly decreased sleep disturbance and increased sleep quality (Carlson & Garland, 2005). It looks like there might be a positive correlation between stress and sleep problems - indicating that sleep might be looked at as an indirect measure of stress. This factor should therefore receive greater attention in stress and weight related research.

Sleep as a Concept

Sleep plays a vital role to our overall physical and mental health, in many ways, which we are just beginning to understand. Similarly to stress, it is a complex phenomenon, and the

underlying mechanisms and functions are not clearly understood yet. There is little doubt that we all require sleep, however; researchers are not sure on why that is. According to National Sleep Foundation (2012), referred to as NSF, most experts supports the following sleep recommendations: school-age children (5-10 years) need 10-11 hours of sleep daily; teens (10-17 years) need 8.5-9 hours; and adults need 7-9 hours. Nonetheless, sleep is highly individual, and health and lifestyle impact the amount of sleep needed. The sleep phenomena also appears largely invariant from one society to another. A cross-cultural survey from 10 different countries, including about 35,000 participants, did not find much variance in sleep patterns (Soldatos, Allaert, Ohta, & Dikeos, 2005).

Consequences of Inadequate sleep

In a recent review by Banks & Dinges (2007), reduced sleep in adults - on a short term basis, were related to many negative health effects. The findings included sympathetic nervous activation, elevated blood pressure, reduced glucose tolerance, hormonal changes (reduced leptin and increased grehlin), altered inflammatory responses, weight gain, and even increased risk of mortality. Additionally, vital behavior of daily functions declined, such as; cognition, memory and mood. Furthermore, the Centers for Disease Control (CDC), found that adults in the United States who slept less than six hours were more likely to engage in risky health behaviors, compared to adults who slept 7 to 8 hours (Schoenborn & Adams, 2010). In Australia, a new study with approximately twenty thousand young adults participating (aged 17-24), found evidence that shorter sleep duration were linearly associated with prevalent and continuous psychological distress (Glozier et al., 2010).

Sleep and Weight Gain

Inadequate amounts of sleep are thus related to many serious health problems and also in fact related to weight gain (Patel & Hu, 2008; Sorensen & Ursin, 2001). Cappuccio and fellow researchers (2008) recently conducted a large population based meta- analysis, which included cross-sectional studies from around the world. Their results suggested that participants with short sleep duration were persistently at an increased risk of obesity. The study included both children and adults (aged 2-102). However, they did not find evidence of temporal sequel in longitudinal designs. In Norway, research among school-age children (aged 10-12), have found similar results, although high BMI status were related to both short and long sleep duration (Danielsen,

Pallesen, Stormark, Nordhus, & Bjorvatn, 2010). In relation to sleep quality, there is only one study found on this age group that addresses this nationally. A decade ago, Sorensen and Ursin (2001) found that adolescents in a small Norwegian sample (22) were unsatisfied with the amount of sleep received (7.3 hours), and this is also below age recommended levels according to the National Sleep Foundation (2012). The questions still remains as to whether short sleep duration and increased BMI pertains to Norwegian adolescents' as well.

Multiple studies and reviews have shown an inverse association between short sleep duration and increased BMI. Other studies have reported a U-shaped curvilinear relationship between short and long sleep duration (Danielsen et al., 2010). Most of these studies are done using cross-sectional designs and self reports (Schoenborn & Adams, 2010; Van Cauter & Knutson, 2008)

Patel and Hu (2008) proposed a causal pathway theory linking sleep with weight gain. These explanations are based on a literature review on the subject matter and appear simple but congruent with evidence so far.

1. Consistent sleep deprivation can result in fatigue, which may lead to physical inactivity.
2. Reduced time sleeping allows for more time to eat.
3. Sleep deprivation may affect caloric consumption through the neuroendocrines system. Possible explanations for this effect proposed by the researchers include decreased levels of the hormone leptin or increased levels ghrelin hormone.

Possible Causal Pathways between Stress and Overweight Development

Some components of the neuroendocrine pathways of the stress-system are also involved in the food intake regulation system (Valassi, Scacchi, & Cavagnini, 2008). This interaction could account for a neuroendocrine connection between stress and food intake regulation. Several studies have established an association between stress and eating behavior (Crowther, Snaftner, Bonifazi, & Shepherd, 2001; Freeman & Gil, 2004; Wolff, Crosby, Roberts, & Wittrock, 2000). However, is a higher level of stress connected to higher energy intake? Willenbring, Levine, & Morley (1986) and Weinstein, Shide, & Rolls (1997) found through self-reports that stress affects eating, although in different ways for different people:

Restrained versus Non-Restrained Eaters

Approximately half of the people eat more when under stress and half eat less. A comparable result was found by Oliver and Wardle (1999), with 39% of their participants reporting a hypophagic (under eating) response to stress and 42% reporting a hyperphagic (over eating) responses. A substantial minority (19%) reported no change in eating behavior. This inconsistency could reflect random variation, yet one longitudinal study by Stone & Brownell (1994), proposed that dietary responses to stress, might be a stable individual trait – thus suggesting that some people are consistently hyperphagic and others consistently hypophagic. For example, restrained eaters typically show higher intake in high stress conditions than in low stress conditions, while non-restrained eaters either eat the same amount or less in the high stress situation (Heatherton, Herman, & Polivy, 1991; Oliver & Wardle, 1999; Polivy, Herman, & McFarlane, 1994; Schotte, Cools, & McNally, 1990).

Stress Reactivity and Coping Style

As mentioned earlier, reactivity to stress is based on individual traits and partially based on cognitive reactions. A possible explanation distinguishing over-eaters from under-eaters may be stress reactivity. Specifically, high cortisol reactivity may lead to eating in response to stress, given the relationship between cortisol with both stress, and mechanisms affecting hunger. (Tataranni et al., 1996). Epel, Lapidus, McEwen, and Brownell (2001), found that women who were high cortisol reactors to stress ate more food when recovering from stress than women who were low reactors. During the rest day, high reactors tended to eat less, and low reactors tended to eat more, eliminating the difference between the two groups. Nevertheless, high cortisol reactors tended to consume more sweet foods than low reactors, across days. Similarly, eating behavior when stressed may also depend upon the coping style one normally use, Sulkowski, Dempsey, and Dempsey (2011), researched the associations between stress coping and binge eating in female college students and found that emotion-focused and avoidant coping style were positively associated with stress and binge eating. No association was found between stress, rational or detached coping, and binge eating.

Stress and a Higher Fat Diet

Seemingly stress may lead to increased food intake in general, but does it also affect the quality of the food intake? Ng and Jeffrey (2003) and McCann, Warnick, & Knopp (1990) have found that greater perceived stress is positively associated with a higher fat diet. Also, Oliver and

Wardle (1999) found that under stress circumstances, the foods most frequently reported as being eaten in greater quantity were sweets and chocolate (70%), cakes and biscuits (60%), and savory snacks (48%). Foods least likely to be eaten in greater quantity were fruit and vegetables (19%) and meat and fish (9%). Notably, in both hypophagic and hyperphagic responders, stress was associated with a shift toward consumption of more pleasant foods, and 73% of the participants reported an increase in snacking. The findings are congruent with other studies and also on adolescent populations (Cartwright et al., 2003a; Oliver & Wardle, 1999; Torres & Nowson, 2007). Nevertheless, the research within this field have yielded mixed results, for instance Epel et al., (2001) found that stress did not alter consumption of a range of foods, including high- and low-fat sweet foods, compared with the non-stressed condition. It is also important to note that there are limitations in studies examining stress-related eating behavior, including less than optimal method to measure stress levels (Ng & Jeffery, 2003) and small samples (McCann et al., 1990).

Stress and Physical Activity

The relationship between chronic stress and energy expenditure needs to be considered when investigating the etiology of overweight and obesity. There is evidence for a negative association between perceived stress and levels of physical activity in adolescents (Norris, Carroll, & Cochrane, 1992). In particular, experiencing weight bias stress has shown to have a negative effect on physical activity among school children. In a review article by Puhl and Latner (2007), three studies were cited in which perception of weight bias were negatively related to, or lead to avoidance of - physical education classes. On the other hand, physical activity is considered a protective factor against the effects of stress on obesity in adolescents (Haugland, Wold, & Torsheim, 2003; Yin, Davis, Moore, & Treiber, 2005), making this subject especially important to further investigate.

Stress and Overweight

There exists an association between stress and eating habits, but does this mean there exists one between stress and overweight? If stress causes some individuals to consume food in excess of requirements, this may terminate in weight gain, and researchers have hypothesized that stress may be an etiologic factor in the development of obesity (De Vriendt et al., 2009; Gunnar & Cheatham, 2003; Muennig & Bench, 2009). Björntorp (2001), reviewed the

underlying physiological mechanisms of the potential connection between stress and abdominal obesity and concluded that elevated cortisol secretion, caused by stress, might lead to visceral fat accumulation. He also hypothesized that glucocorticoids may disrupt the food intake regulation in humans by stimulating the neuropeptide Y system (food intake stimulation) and moderating the effect of the leptin system (food intake reduction). Thus, increased cortisol secretion caused by chronic stress may result in a long-term increased energy intake and fat accumulation. This is also congruent with a recent epidemiological review of adolescence. Also here, the data provided evidence of a positive relationship between stress and weight gain, more specifically – obesity (De Vriendt et al., 2009). Despite the fact that cortisol as a biomarker of stress often is linked to obesity related parameters, the connection between obesity and cortisol is not clear cut, Travison, O'Donnell, Araujo, Matsumoto, and Mckinlay (2007), linked cortisol with the etiology of obesity in men, and their results suggested a weak connection between absolute levels of serum cortisol and measures of adiposity. However, a similar study found no significant correlation in overweight Latino children and adolescents, aged 8 – 13 years (Weigensberg, Toledo-Corral, & Goran, 2008).

Stress, Energy Intake and Obesity

If stress-induced eating were contributing to the development of obesity, it would be probable that obese individuals would consume more food in response to stress compared with slim individuals. Slochower, Kaplan, and Mann (1981) for instance, found an increase in food-consumption in obese individuals. Also, a large cross-sectional study found a slight association between work stress and BMI (Kouvonen, Kivimaki, Cox, Cox, & Vahtera, 2005). Similarly, Kivimaki et al.,(2006) found that work stress increased the likelihood of weight gain in those with higher BMI but, was likely to predict weight loss in lean individuals, yet this observed bidirectional effect was seen only in men and not in women. Several other studies have found positive associations between stress induced eating and overweight, especially in men.

Gender Differences

There might be a gender-specific response to stress; Laitinen, Ek, and Sovio (2002) revealed that stress related eating (defined as trying to make oneself feel better by eating or drinking in a stressful situation) was significantly associated with obesity, but only in women and not in men. Maybe women are more likely to use food to deal with stress, whereas men

might cope with stress through other incitements, such as alcohol consumption (Mehlum, 1999) or cigarette smoking (Conway, Vickers, Ward, & Rahe, 1981). In addition, Cartwright et al. found that women, restrained eaters and overweight or obese individuals tend to consume a greater quantity of food when stressed, compared to men, non-restrained eaters and normal weight individuals (Cartwright et al., 2003b). Oliver and Wardle (1999) also found through their questionnaire that women were slightly more likely than men to report a hyperphagic response to stress, and slightly less likely to report no change, while the proportions reporting hypophagic responses were very similar between women and men (respectively 40% and 37%). However, the gender difference in intake in response to stress, just failed to reach statistical significance. Furthermore, the effect of stressful life events over a 6 months period on change in BMI was studied in men and women who were classified as high (respond to stress by eating more) or low (respond to stress by eating less) emotional eaters. Only male, high emotional eaters who reported more than three stressful life events had an increase in weight within 6 months (Van Strien, Rookus, Bergers, Frijters, & Defares, 1986) The subject matter on whether there is a gender difference in the response to stress is hence disputed.

Puberty may be a vulnerable and critical phase for many teens. Not only are adolescents' experiencing noteworthy developmental changes physically and mentally at this time, additional influences of stress may exacerbate occurrences taking place in this period, and eventually lead to unhealthy weight gain. Weight related research on this age group is scarce and to our knowledge, no other studies with a longitudinal perspective, has investigated the effects of stress and sleep on weight status on youth in Oslo. Thus, the present study seeks to generate important information on the relationship between stress and sleep on the development of overweight adolescents'. More specifically, explore the influence of "Life Stress", "Daily Hassles", "Violence", and "Sleep Duration", in relation to BMI status on youth living in Oslo.

AIMS – Research Questions to be Approached in this Study

The aims of this research project are both descriptive and inferential in nature. Based on the above we formulate the following research questions:

1. Are there any gender differences between the stress variables and weight categories?

2. Does stress measured as Life Stress, Sleep Duration, Violence and Daily Hassles predict the level of body weight three years later?
3. Does stress measured as Life Stress, Sleep Duration, Violence and Daily Hassles predict the probability of developing overweight three years later?
4. Are there any changes in the stress variables over a three-year period and if so; how does any possible changes affect body weight?

Methods

Norwegian Social Research Institute (NOVA) conducted a quantitative survey with a longitudinal design on 3 different test points with approximately a year and a half in between each testing point. The study was named Young in Oslo (LUNO) and targeted adolescents' residing in Oslo Municipality, Kristinn Hegna at NOVA is the project manager. The Regional Ethics Committee and the Data Inspectorate has approved the study. We, authors of this paper, were provided with the results of Test 1, Test 2, and Test 3 (hereafter referred to as T1 – T3), on an anonymous statistical computer file, in SPSS format, which contained the organized variables and its data.

Participants and Procedure

The total Oslo population consists of about 4680 students in 9th grade, in 2006. Out of these, 4022 (85.9%) students were asked to participate in LUNO. The number reflects that a few schools had declined and specialty schools were not asked to participate. Participation consent forms were received from 2416 students, which constitute 60 percent of those originally asked.

Response rates

- In T1 (2006): the response rate was 2328 (1060 boys and 1240 girls, range 13-15 years of age, and mean = 13.9). Of these, 82 % of the students (n = 1913), responded in T2, and 1748 (75,1%) responded in T3. There were 1569 students whom responded on all three times (67,4%).
- In T2 (2008): the response rate was 2029 (928 boys and 1101 girls, range 14 - 17 years of age, and mean = 15.3).
- In T3 (2009): the response rate was 1865 (820 boys and 1032 girls, range 16 -19 years of age, and mean = 17.6). To note, of these, 27 (1,4%) were new respondents.

Of those who originally withdrew from T1, the research team managed to get almost half to come back and respond in T3. Of those who had responded in both T1 and T2, 82,1% also responded in T3. The present study is based on all participants that responded in T3 (in addition to T1 and T2).

In general, NOVA provided special assistance or training if needed. To participate in the study, an informed consent form needed to be signed by both student and parent/guardian. The informed consent form, described the length of the project, study intention, and implementation timeframe. It also highlighted that participation is voluntary, that only restricted individuals will have access to the raw data and perform necessary data analysis, and that all data is stored and handled according to guidelines. As well as that any publishing of the survey results will not in any way reveal individual responses. Every student also received a unique id number that linked their names, addresses, school, and birth numbers. The teachers were given responsibility to record the students' code after questionnaire completion.

Early on, a pilot study was implemented to assess: 1) the survey questions and 2) the use of electronic questionnaires versus paper questionnaires.

T1: General information about the survey was provided to the students and parents/guardians. The questionnaires were in paper format and made for self-completion. After completion, the signed consent forms along with the questionnaires were sent to NOVA.

T2: In T2 the survey was also available in electronic format, one was through email; the other was through a URL – address. A letter and an email with survey information and the procedures were sent out to all school contact individuals. The participants' names and related id numbers were enclosed. Again, information about the survey was provided to students and their parents/guardians in a brochure sent by mail. Several reminders were frequently sent out up towards the end of the data collection time, to increase the response rate.

T3: A letter was sent out to all the principals in the different high schools, asking for approval of T3 implementation. A brochure was sent out by mail to all participants, with information about the third questionnaire and some tentative results from T1 and T2, to illustrate what the survey results may be used for. The questionnaires were in electronic format and available through an email link or an alternate solution - if there was no email address available.

Design and Material

LUNO, is a longitudinal survey with presently three data collection times (fall 2006; spring 2008; and fall 2009). In T1 all the paper questionnaires were optically scanned. In T2, some new questions were added and some old ones were left out. Additionally, the questionnaires in T2 and T3 were in electronic format.

The questions that were asked in this study reflect the particular focus on risk factors and protective factors as predictor variables, and different forms of mental health, school- and work choice and social adjustment and integration as outcome (dependent) variables. In addition, there was a specific focus on youth as immigrants and/or with disabilities and their experiences. Many of the variables were also taken from NOVA's other big youth studies, and consequently allows for comparison between the data sets.

Dependent Variable

Body Mass Index (BMI), the dependent variable, is measured by self-reported height and weigh (kg/m^2). To this date, there are no agreements on which universal type of measurement to employ when defining overweight/obesity. Defining overweight and obesity amongst children and adolescents have shown to be problematical endeavor, due to the fact that their height is continuously changing, and so does their body proportions. Despite inherent weaknesses, the usage of BMI appears to be increasingly accepted as a valid measure of weight status among adults, as well as on children and adolescents. Although an elevated BMI among adults are consistently linked with increased mortality, similar research on children and adolescents are sparse. A recent finding from a British cohort from 1946 – until 60 years of age, or death, did find evidence of premature mortality risks associated with increased BMI (Strand, Kuh, Shah, Guralnik, & Hardy, 2012). Bjorge, Engeland, Tverdal, and Smith (2008), have found comparable results on Norwegian teenagers. More importantly, according to Flegal and Ogden (2011), it is essential to note that the cut-off points in BMI, indicates an increased risk of negative health outcome, and are not a diagnostic criterion in itself. The BMI is a mainly a tool which assists in identifying weight changes.

Adolescents in our study were grouped according to age and sex cutoff values from United States Centers for Disease Control and Prevention as displayed in the table below (2012). The reference group in this tool, aged 2-20, was based on 5 National surveys. Through statistical

analyses, averages and deviation categories from a diverse population were calculated and used as a basis for the different categories (Kuczmarski et al., 2002).

Table 1

BMI cut-off values for boys and girls 12 – 18 years of age

		Underweight	*Normal*	*Overweight*	*Obese*
Girls					
	Age 12	≤14.7	14.8 – 21.6	21.7 – 26.0	≥26.7
	Age 13	≤15.2	15.3 – 22.5	22.6 – 27.7	≥27.8
	Age 14	≤15.7	15.8 – 23.2	23.3 – 28.5	≥28.6
	Age 15	≤16.2	16.3 – 23.8	23.9 – 29.0	≥29.1
	Age 16	≤16.7	16.8 – 24.3	24.4 – 29.3	≥29.4
	Age 17	≤17.0	17.1 – 24.6	24.7 – 29.6	≥29.7
	Age 18	≤18.4	18.5 – 24.9	25.0 – 29.9	≥30.0
Boys					
	Age 12	≤14.9	15.0 – 21.1	21.2 – 25.9	≥26.0
	Age 13	≤15.4	15.5 – 21.8	21.9 – 26.7	≥26.8
	Age 14	≤15.9	16.0 – 22.5	22.6 – 27.5	≥27.6
	Age 15	≤16.4	16.5 – 23.2	23.3 – 28.2	≥28.3
	Age 16	≤17.0	17.1 – 23.8	23.9 – 28.8	≥28.9
	Age 17	≤17.6	17.7 – 24.4	24.5 – 29.3	≥29.4
	Age 18	≤18.4	18.5 – 24.9	25.0 – 29.9	≥30.0

To be noted, the BMI variable will be analyzed in either as a dichotomous or continuous variable. In the dichotomous form, the BMI is split into those that are normal weight throughout all test points (1), versus those that are normal weight in T1 and overweight in T3 (2).

Independent Variables

Early Adolescent Stress Questionnaire (EASQ), referred to as Life Stress in this study, measures events that are stressful. It has been translated and adjusted to fit Norwegian youth (Sund, Larsson, & Wichstrøm, 2003). The instruments consist of 32 statements. It measures stress periods through one's lifetime and the response alternatives are either "Yes" or "No". Examples of statements used are: "Your parents got separated/divorced", "Someone you cared for died (relative, good friend)", and "One of your friends were in serious trouble". The scale is summed so that a higher score equals higher number of stressful life events, and it was only used in T1.

The Urban Hassles Index (Miller & Townsend, 2005), is meant to provide a picture of additional everyday stressors related to city living. The scale is made up of parts of the index and consists of 15 questions, regarding experiences happening during the last two weeks and four of these regarding the local community where one lives. The questions have been adjusted to fit

Norwegian conditions. Examples are: “Have you experienced that a drug abuser has begged you for money?”, “How often have you been afraid because the area you live in is not safe?”, and “How often have you experienced friends pushing you into fights?”. All possible types of responses provided are based on a 4-point Likert scale (coded 1 “never”, 2 “sometimes”, 3 “often”, and 4 “very often”). An exploratory principal component factor analyses were conducted. The aim was, to have sets of items that showed a pattern of responses that made it possible to have them in a homogenous index (See Appendix B for details). The result was three strong factors with items that theoretically constituted Daily Hassles, a) Hassle Bullying, b) Hassle Criminal Encounters, and c) Hassle Local Community, consisting of the four questions regarding local community. In the present analyses we used the three factors in each of the samples as basis for index construction. The index is made up by taking the total sum and dividing it by the number of items, and a higher score on this index suggests more experiences with Daily Hassles. Not all of the 15 items were used in T2, see appendix for information about which elements were removed.

Violence Exposure**,** are questions based upon experienced Violence, and they were taken from the study; Young in Norway 2002. They consist of four statements of Violence experiences from the last 12 months. The statements used to assess Violence is: “I have been exposed to threats of violence”; “I have been beaten without visible markings”; “I have been wounded or injured because of violence without the need of medical treatment”; and “I have been injured so strongly because of violence that I needed medical treatment”. Response alternatives include (1) “many times”; (2) “a few times”; (3) “one time”; and (4) “never”. The values were reversed and the total sum was divided by four, now a higher score on this index suggests more experiences with Violence.

Sleep Duration: the Competency center for the study of sleep problems (related to the University of Bergen) developed a series of sleep revolving questions. Only the question which pertained to Sleep Duration on weekdays was used in analysis and it was formulated as follows: “How much sleep do you generally get on ordinary weekdays?” The participants were to fill in the amount of hours of sleep received, and obviously, a higher number indicate longer sleep duration.

Differential predictor variables

Since the predictor variables, except for the Life Stress variable, are presented in both T1 and T2, the discrepancy between these variables on the different test times will also be used as predictors in the analyses. That is, the difference between reported level of the Hassles instruments on T1 and T2, will be used as predictor variables, and the same hold true for Violence exposure and Sleep Duration. To get the differentiation between T1 and T2, the mean on T1 was subtracted from the mean on T2 – controlling for the reduced amount of items in T2. A positive value indicates an increase in stress level (Hassles and Violence) and a decrease in Sleep Duration.

Attrition

Several measures were performed in order to evaluate attrition and data representativeness. The first step were done to explore any gender differences in the missing data between the three test points, specifically, missing data between Test point 1 and Test point 3. The first variable compared participants from only T1/T2, with those that are still participating at T3. The second variable compares those who have participated only at T1, with those who are still there at T3. A Crosstabulation using Chi squared test compared Variable 1: participated in T1/or T1 and T2, with Group 2: Participated in T1 and T3/or T1, T2 and T3, then split on gender, X^2 (1, n =2300) =8.06, p <.05. The results revealed that there was a significant gender difference, such that; more boys went missing between the test points. In other words, more girls participated in the study as a total. Furthermore, the next step examines changes on missing data with all the stress variables in relation to the test points.

Independent t-tests revealed a considerably differences in the average scores for Hassle: Violence exposure in those who only participated in T1 (M =1.34, SD =.60) and those who participated in T1 and T3/or T1, T2 and T3 test points; (M =1.23, SD =.45) t (798) = 4.73, p < .001. Additionally, boys whom had dropped out after T1 had a significantly higher level of Life Stress (M =8.7, SD =5.9) than those that remained in the study until T3 (M =7.4, SD =4.8; t (770) = 2.04, p <.05. The same tendency were found among girls, however, the difference was not significant (only T1: M = 8.8, SD = 5.2, participated in T1-T3: M = 7.8, SD = 4.6; t (938) = 1.95, p = .051). These results suggest that boys have a higher mean in relation to Violence exposure on the earliest test point, compared to the boys that remain in the study. However, this

selection drop-out bias is controlled by conducting analysis split on gender. Furthermore, it is likely that we may have underestimated the effect between Violence exposure and Life Stress on weight gain, because those that are exposed to higher level of Violence and Life Stress, the boys, have also left the study early on.

Statistical Analysis

The Statistical Package for the Social Sciences (SPSS 19) was used for analysis. Since every item in each instrument have the same weight, indexes were constructed by adding the items and then divided by the number of items. A Crosstabulation analysis will be used to examine gender differences in BMI and to assess for any BMI changes over the test points. Subsequently, correlation coefficients will be computed to assess a bi-variate relationship between the stress variables and BMI. First, to investigate the relationship between each of the stress variables in T1 and BMI on T3, a stepwise backward linear regression analysis will be conducted. The stepwise backward regression analysis is beneficial when one does not have a theoretical foundation for any prediction(s) and rather center ones attention on exploring possible relationships. The method starts out with all the stress variables in a model and stepwise - removes the variable which has the lowest partial association, given the other predictors in the equation. At the end, a coefficient is assigned to each stress variable, and it signifies the strength of each one in predicting elevated BMI in T3. The next step is a backward stepwise logistic regression. It investigates the relation between BMI and stress by focusing on the participants that originally were normal weight and went on to develop overweight in T3. Due to the adverse health effects associated with increased body weight, the main purposes here is to see how many participants have developed excessive weight gain in such a short period (3 years), and thus; are now at risk. Additionally, a paired sampled t-test will be conducted to evaluate possible stress changes from T1 (2006) to T2 (2008) among girls and boys. Finally, a linear regression analysis will be conducted to see if changes in any of the stress variables from T1 to T2 are significantly related to increase in BMI on T3.

Results

The study explored different stress variables influences on BMI status among adolescents in Oslo. The first section contains descriptive data on the participants and the diverse variables.

A corresponding inferential section follows with analyses testing the relationships between the stress variables and BMI.

Table 2

Socio-demographic Variables at T1 separated by gender

		Gender							
		Girls				Boys			
		N	*%*	*M*	*SD*	*N*	*%*	*M*	*SD*
Gender		1240	53.9			1060	46.1		
Age		948		13.9		763		13.9	
Born in Norway :	Yes	864	88.4			696	89.8		
	No	92	11.6			70	10.2		
Parents Marital status:									
	Married (M)	604	63.3			460	59.3		
	Cohabiting (C)	48	5.2			51	6.1		
	Divorced/Separated	257	28.0			224	32.7		
	Never M or C	3	3.5			13	1.9		
*District of Upbringing:									
	Inner East	124	13.9			90	11.9		
	Inner West	73	7.9			48	6.7		
	Outer West	331	33.3			269	34.5		
	Outer-East	420	44.1			345	46		
	Not Oslo	8	0.7			4	0.8		
*Social Class:									
	Upper service class	315	30.9			273	31.8		
	Lower service class	352	34.6			293	34.1		
	Clerk	69	6.8			49	5.7		
	Self employed	2	0.2			1	0.1		
	Skilled worker	68	6.7			57	6.6		
	Unskilled worker	91	8.9			64	7.5		
	Unknown/Un-coded	121	11.9			122	14.2		
BMI		878		19.4 (11-55)	3.6	722		19.6 (12-57)	3.3

*District of upbringing: Inner East Side (Sagene, Grünerløkka), Inner West Side (Frogner, St. Hanshaugen), Outer West Side (Ullern, Vestre Aker, Nordre Aker, Sentrum, Nordstrand), Outer East Side (Alna, Bjerke, Østensjø, Stovner, Grorud, Søndre Nordstrand).
*Social Class is based on Ericsson - Goldthorpe classification system and the data stems from the fathers' occupational status (mother if missing T2).

The table represents the distribution of some of the socio-demographic variables taken at Time 1. There was a relatively equal representation between girls and boys, respectively almost 54% and 46%. The large majority was born in Norway, close to 90%. However, this does not include the distinction between second or third generation foreigners. Also, over half of the students came from home with married parents, and close to 1/3 had separated/divorced homes.

Over 30% of both genders were raised on the outer west side of Oslo, over 40% were raised on the outer east side, and less than 1% was not brought up in Oslo. The majority of students reported that their fathers worked in “upper” or “lower” service areas and close to 10% had checked unknown/un-coded. Additionally, graphical representations, specifically - boxplots, were utilized to view dispersion and identify extreme BMI scores. BMI scores below 10 and above 60 were treated as potential outliers and removed from the data before analyses. The average BMI were 19.4 on girls and 19.6 on boys.

The relationship between the socio-demographic variables and body weight, was further examined in T1, and variance analysis (ANOVA), found that girls from a section of the Outer East Side (Alna, Bjerke, Østensjø) weighed significantly more (M = 20.0, SD = 4.5, n = 237) than girls from Inner West Side (Frogner, St. Hanshaugen) (M = 18.5, SD = 2.3, n = 89, F = 3.30, p <.01). There was no difference between BMI and district of upbringing with boys. On the other hand, an association was observed in boys between lower social class in T1 and a higher BMI three years later (r =.15, p <.05). No effects were found among girls and social class.

The question as to whether there is gender differences between BMI categories naturally arose, and thus; a cross-tabulation analysis was conducted in table 3.

Table 3

Contingency table: BMI Categories based on Girls and Boys in T1, T2 and T3

BMI Categories		Gender					
		Girls		Boys		Of Total	
		n	*%*	*N*	*%*	*N*	*%*
T1: BMI	Underweight	44	2.8	37	2.4	81	5.2
	Normal	747	86.7	592	83.4	1339	85.2
	Overweight	54	6.3	65	9.2	119	7.6
	Obese	17	2.0	16	2.3	33	2.1
Total		862	100	710	100	1572	100
T2: BMI	Underweight	43	5.4	23	3.6	66	4.6
	Normal	694	87.1	528	83.5	1222	85.5
	Overweight	51	6.4	67	10.6	118	8.3
	Obese	9	1.1	14	2.2	23	1.6
Total		797	100	632	100	1429	100
T3: BMI	Underweight	37	4.2	32	4.6	69	4.4
	Normal	766	87.0	562	80.4	1328	84.1
	Overweight	65	7.4	85	12.2	150	9.5
	Obese	12	1.4	20	2.9	32	2.0
Total		880	100	699	100	1579	100

A Chi square test of independence was performed to determine if the BMI categories were equal among both genders. In T1, the BMI categories were equally distributed among boys and girls T1: BMI Categories and Gender, χ^2 (3, n =1572) =.41, p = .061. On the other hand, The BMI categories were not equally distributed among boys and girls in T2, BMI Categories and Gender, χ^2(3, n =1429) =12.98, p <.05. And in T3: BMI Categories and Gender, χ^2 (3, n =1579) =15.83, p <.001. The results showed that boys are significantly more prominent in higher weight categories than girls. Subsequently, the next section looks at any noteworthy changes within the BMI categories that occurred over the three years, and this was also tested using crosstabulation (table 4).

Table 4.

Change in weight status among boys, girls, and all - from T1 (2006) to T3 (2009)

	Weight status (2009)					
	Normal weight		Overweight		Obesity	
All Weight (2006)	*n*	*%*	*n*	*%*	*n*	*%*
Normal weight	1218	93.5	73	5.6	11	0.8
Overweight	43	42.2	52	51.0	7	6.9
Obesity	13	41.9	7	22.6	11	35.5
Total	1274	88.8	132	9.2	29	2.0
Girls Weight (2006)	N	%	n	%	n	%
Normal weight	692	94.8	34	4.7	4	0.5
Overweight	22	52.4	19	45.2	1	2.4
Obesity	7	43.8	3	18.8	6	37.5
Total	721	915	56	7.1	11	1.4
Boys Weight (2006)	n	%	N	%	n	%
Normal weight	526	92.0	39	6.8	7	1.2
Overweight	21	35.0	33	55.0	6	10.0
Obesity	13	41.9	7	22.6	11	35.5
Total	553	85.5	76	11.7	18	2.8

Table 4 displays changes in weight status from T1 (2006) to T3 (2009) split on genders. The category of underweight is collapsed with normal weight in this analysis.

A Cramer's V test was performed to determine if any significant BMI changes had occurred from Test 1 (2006) to Test 3 (2009) among both genders. Significant weight changes were seen on the group as a total: Cramer's V (n =1435) =.39, p = <.001, among girls: Cramer's V (n =788) =.41, p =<.001, and also among the boys: Cramer's V (n =647) =.39, p = <.001. Within the Normal weight category, boys have a higher likelihood of developing overweigh and obese status and a higher percentage of girls are considered normal weight three years later. Next, of those who are already overweight in T1, the boys are less likely to become normal weight, and consequently, they have an increased tendency of staying overweight and becoming obese compared to girls. In contrast, the weight category obese shows that boys are less likely to stay obese (in T3) if considered obese in T1 compared to girls, respectively 33.8 % versus 37.5 %, however, they have a higher likelihood of changing to overweight status, whereas girls, have a higher chance of becoming normal weight. There were also differences between BMI and gender on all levels. For example, BMI at T3 for girls (M = 19.99, SD = 3.01) and for boys (M = 21.93, SD = 3.40), t (1407) = 5.95, p <.001.

The previous tables gave information about the participant group, and differences, as well as changes in BMI levels - within and - between the two sex groups. The following tables will explore various analyses regarding the predictor variables' relation to the dependent variable.

Table 5

Correlations, Means, and Standard Deviations of Major Stress Variables at T1 and BMI at T1 and T3 for girls (lower left diagonal) and boys (upper right diagonal).

	Girls		LS	V	SD	HB	HCE	HLC	BMI1	BMI3	Boys	
Items	*n*	*M*									*n*	*M*
Life Stress	841	1.76 (0.14)	-	.46**	-.23**	.32**	.44**	.32**	.07	-.01	681	1.77 (0.15)
Violence exp.	966	1.13 (0.31)	.39**	-	-.10**	.30**	.43**	.32**	.07	-.06	775	1.36 (0.55)
Sleep Duration	936	1.66 (0.70)	-.08**	-.08**	-	.03	-.04**	-.03**	-.20**	-.12**	742	1.56 (0.65)
Hassle Bullying.	967	1.15 (0.24)	.30**	.19**	.02	-	.29**	.36**	-.00	-.01	777	1.19 (0.32)
Hassle CE	967	1.32 (0.32)	.44**	.37**	.10**	.33**	-	.31**	-.09*	-.03	777	1.34 (0.37)
Hassle Local C	969	1.27 (0.36)	.29**	.21**	-.00	.31**	.20**	-	.06	.01	777	1.27 (0.36)
BMI 1	885	19.41 (3.57)	.10**	.13**	-.01	.08*	.15**	.08*	-	.43**	731	19.64 (3.32)
BMI 3	896	20.96 (3.01)	.09*	.10**	-.01	.00	.06	.03	.50**	-	707	21.95 (0.37)

** = p = < .01, * = p = < .05. Standard deviation is within the parentheses below the means. Note. LS = Life Stress; V = Violence exposure; SD = Sleep Duration; HB = Hassle: Bullying; HCE = Hassle: Criminal Encounters; HLC = Hassle: Local Community; BMI 1 = Body Mass Index at T1, BMI 3 = Body Mass Index at T3.

A Pearson product – moment correlation coefficient was computed to assess the relationship between the above stress variables on T1, and BMI - on T1- and T3. Overall, there were small to medium positive relations between all predictor variables for both girls and boys. This indicates, that when one experiences stress on a daily basis, one has a greater chance of also experiencing being threatened or having sleep problems etc. Especially interesting is the strong medium correlations between Violence and Life Stress in both boys and girls, suggesting that experiencing stressful life events equals a greater chance to also experience for example being threatened or beaten. Also, there are gender differences in the associations between Life Stress and Sleep Duration, for girls the correlation is minor whereas for boys it is small to medium. This indicates that Sleep Duration is closer connected to stress for boys than for girls

There was a medium to strong positive correlation between BMI on T1 and BMI on T3 for both girls and boys, suggesting a relative stability in body weight over three years. There were weak positive correlations between the predictor variables and BMI on T1 and T3 for girls, suggesting a weak connection between experienced stress and higher weight. The correlation between stress variables and BMI was equally weak on T1 and T3. For boys, the cross sectional correlation between the stress variables on T1 and BMI on T1 was weakly positive, except for the Hassle: Bullying, which had no correlation. Sleep Duration, had the strongest relation to BMI for boys, indicating that the less sleep length boys had on weekdays, the higher the BMI was.

A stepwise backward linear regression analysis was employed to explore for linearity relations between the stress predictors and BMI in T3, see table 6.

Table 6

Summary of Multiple Linear Backwards Regression Analysis Predicting BMI at T3

		Girls: N = 748					Boys: N =591	
Predictor variable	R^2	β	*T*	*p*	*Predictor variable*	R^2	β	*t*	*p*
Model 1	.01				Model 1	.02			
Life Stress		.06	1.50	.13	Life Stress		-.00	-.03	.98
Violence		.03	.70	.51	Violence		-.07	-1.50	.14
Hassle LC		.03	.65	.43	Hassle LC		.06	1.17	.24
Hassle CE		.03	.80	.15	Hassle CE		-.04	-.70	.48
Hassle B		-.06	-1.43	.78	Hassle B		.02	.39	.69
Sleep Duration		-.01	-.25	.80	Sleep Duration		-.13	-3.11	.00*
Model 2	.01				Model 2	.02			
Life Stress		.07	1.51	.13	Violence		-.07	-1.54	.13
Violence		.03	.71	.48	Hassle LC		.06	1.18	.24
Hassle LC		.03	.65	.52	Hassle CE		-.04	-.74	.46
Hassle CE		.03	.81	.42	Hassle B		.02	.40	.69
Hassle B		-.06	-1.44	.15	Sleep Duration		-.13	-3.11	.00*
Model 3	.01				Model 3	.02			
Life Stress		.07	1.63	.11	Violence		-.07	-1.50	.14
Violence		.03	.78	.44	Hassle LC		.06	1.40	.18
Hassle CE		.04	.84	.40	Hassle CE		-.03	-.69	.49
Hassle B		-.05	-1.33	.19	Sleep Duration		-.13	-3.10	.00
Model 4	.01				Model 4	.02			
Life Stress		.08	1.93	.054	Violence		-.08	-1.89	.06
Hassle C		.04	1.00	.32	Hassle LC		.05	1.23	.22
Hassle B		-.05	-1.31	.19	Sleep Duration		1.13	-3.10	.00*
Model 5	.01				Model 5	.02			
Life Stress		.09	2.47	.01*	Violence		-.06	-1.57	.12
Hassle B		-.04	-1.12	.26	Sleep Duration		-.13	-3.08	.00*
Model 6	.01				Model 6	.02			
Life Stress		.08	2.24	.03*	Sleep Duration		-.12	-2.96	.00*

Note: β = standardized beta coefficient, * = significant at p <.05. Hassle LC = Local Community; Hassle C = Criminal Encounters; and Hassle B = Bullying.

The results of this regression indicated that two predictors explained the variance in BMI for respectively girls and boys: Life Stress (controlled for the other stress variables) – significantly predicted an increase in BMI three years later for girls. The more Life Stress the girl

experienced in T1, the higher her BMI was three years later. Sleep Duration (controlled for the other variables) significantly predicted an increase in BMI three years later for boys. The less sleep the boy had on weekdays in T1, the higher his BMI was three years later.

A step further, the question arises as to whether the same predictors differentiate between those that remains normal weight through all the test points (group 1) and those that become overweight (group 2). As seen in table 4, as a total in T1, 1218 participants were considered normal weight, out of these; 84 were overweight/obese in T3. Among girls, 692 were considered normal weight in T1, and in T3, 38 had developed overweight/obese status. In T1, 562 boys were considered normal weight, and in T3, 46 of these were overweight/obese. In order to identify the probability to become overweight 3 years later a logistic regression was conducted, see Table 7.

Table 7

Summary of multiple backwards logistic regression analysis Predicting Change in BMI from T1 to T3 as Criterion (Normal weight T1 – Normal weight T3 (1), Normal weight T1 – Overweight T3 (2).

	Girls				Boys		
Predictor variable	OR	95% CI	*P*	Predictor variable	OR	*95% CI*	*p*
Model 1				Model 1			
Life Stress	1.04	[0.96,1.14]	.33	Life Stress	1.01	[0.92,1.10]	.90
Violence	1.54	[0.52,4.92]	.44	Violence	.81	[0.28,1.74]	.60
Hassle LC	1.33	[0.50,3.55]	.57	Hassle LC	1.47	[0.56,3.89]	.44
Hassle CE	1.29	[0.38,4.34]	.68	Hassle CE	.56	[0.18,1.77]	.33
Hassle B	.92	[0.20,4.20]	.92	Hassle B	1.08	[0.31,3.72]	.91
Sleep Duration	1.01	[0.81,1.27]	.92	Sleep Duration	.86	[0.66,1.13]	.28
Model 2				Model 2			
Life Stress	1.05	[0.96,1.14]	.33	Life Stress	1.01	[0.93,1.11]	.88
Violence	1.53	[0.52,4.50]	.44	Violence	.82	[0.39,1.73]	.60
Hassle LC	1.34	[0.50,3.55]	.56	Hassle LC	1.50	[0.59,3.78]	.40
Hassle CE	1.28	[0.38,4.30]	.39	Hassle CE	.57	[0.18,1.77]	.33
Hassle B	.93	[0.20,4.21]	.92	Sleep Duration	.86	[0.66,1.13]	.28
Model 3				Model 3			
Life Stress	1.04	[0.96,1.14]	.33	Violence	.86	[0.66,1.13]	.28
Violence	1.53	[0.52,4.49]	.44	Hassle LC	.83	[0.40,1.72]	.62
Hassle LC	1.37	[0.41,3.50]	.52	Hassle CE	.58	[0.19,1.73]	.33
Hassle CE	1.27	[0.39,4.16]	.70	Sleep Duration	.86	[0.66,1.13]	.28
Model 4				Model 4			
Life Stress	1.05	[0.96,1.14]	.24	Hassle LC	1.45	[0.59,3.52]	.42
Violence	1.60	[0.56,4.55]	.38	Hassle CE	.53	[0.19,1.47]	.22
Hassle LC	1.33	[0.52,3.44]	.55	Sleep Duration	.87	[0.67,1.13]	.31
Model 5				Model 5			
Life Stress	1.06	[0.98,1.14]	.18	Hassle CE	.59	[0.22,1.59]	.30
Violence	1.65	[0.58,4.56]	.35	Sleep Duration	.87	[0.67,1.13]	.29
Model 6				Model 6			
Life Stress	1.07	[1.00,1.15]	.05*	Hassle CE	.62	[.23,1.66]	.34

Note: * = significant at p <.05; CI = confidence interval; OR = Odds Ratio; Hassle LC = Local Community; Hassle C = Criminal Encounters; and Hassle B = Bullying. Girls (1): N = 594 and (2) N = 31, Boys (1) N = 445 and (2) N = 40.

The results of this regression indicated no significant relationship between the stress variables and the chance for normal weight boys to develop overweight three years later. However, Life Stress significantly predicted the girls' chances to develop overweight three years later. If a normal weight girl experienced Life Stress in T1, there was a significantly greater chance for her to become overweight three years later, given the other stress variables in the equation, albeit; the OR is small. It is to be noted, however, that Life Stress only became significant when Violence exposure was removed in the 5th step.

The next step were to examine if there was any changes in the experience between the first two test points, and by gender, hence, a t-test was implemented to explore this matter, see Table 8.

Table 8

Paired sample t- tests of stress variables from T1, T2, split on gender

	Girls						Boys					
	T1			T2			T1			T2		
Stress	*N*	*M*	*SD*	*M*	*SD*	*T*	*N*	*M*	*SD*	*M*	*SD*	*T*
Hassle LC	1006	1.30	0.34	1.33	0.48	- 4.84***	838	1.27	0.37	1.32	0.50	-2.83*
Hassle C E	1004	1.32	0.32	1.40	0.40	- 6.87***	833	1.34	0.36	1.59	0.58	-12.70***
Hassle B	1004	1.16	0.25	1.13	0.29	3.31***	834	1.18	0.31	1.23	0.45	-2.60*
Violence exp.	999	1.13	0.32	1.20	0.51	- 4.0***	832	1.36	0.54	1.44	0.54	-2.90*
Sleep Duration	933	7.96	1.40	7.36	1.50	10.56***	748	8.15	1.77	7.51	1.84	7.40***

To be noted, Hassles: LC =Local Community, CE = Criminal Encounters, B = Bullying * = p < .05, *** = p < .001. Due to no measurement taken in T2, Life Stress is not included.

The paired-samples t-test found significant change in all the stress variables in this timeframe. The average levels of perceived stress in Oslo students in regards to the Hassle Local Community, Hassle Criminal Encounters, Hassle Bullying, Violence encounters, and Sleep Duration increased among both genders, except Hassle Bullying in girls; which decreased. Overall, these results suggest that the students encounter more stress as they get older.

The effect size (Eta squared) among the girls were small in Hassle: Local Community and Violence encounters = .02, Hassle: Criminal Encounters = 0.04, Hassle Bullying = 0.01, and large in Sleep Duration = 0.10. In boys, the effect size (Eta squared) were small in Hassle Local

Community, Hassle Bullying and Violence encounters = 0.01, moderate in Sleep Duration = 0.07, and a large effect size in Hassle Criminal Encounters = 0.16.

The final question arose as to whether the changes in stress levels between T1 and T2 are related to weight gain.

In other words, does an increase in perceived stress level predict any weight changes in T3? Thus, a multiple stepwise backward linear regression was conducted to explore whether such differences existed, see Table 9.

Table 9

Summary of Multiple Linear Stepwise Backwards Regression Analysis: the Difference between T2 –T1 in Stress Variables on BMI

	Girls: N = 738					Boys: N =563			
Predictor variable	*R²*	*β*	*t*	*p*	*Predictor variable*	*R²*	*β*	*T*	*p*
Model 1	.01				Model 1	.01			
Hassle C E		-.07	-1.44	.15	Hassle C E		.04	.77	.44
Hassle L C		.01	.35	.73	Hassle L C		-.03	-.72	.47
Hassle B		.07	1.66	.09	Hassle B		-.05	-.1.06	.29
Sleep Duration		-.02	-.63	.53	Sleep Duration		.09	2.05	.04*
Violence		-.04	-.99	.32	Violence		.03	.74	.46
Model 2	.01				Model 2	,01			
Hassle C E		-.06	-1.41	.16	Hassle C E		.30	.70	.49
Hassle B		.07	1.73	.08	Hassle B		-.06	-1.27	.21
Sleep Duration		-.02	-.63	.53	Sleep Duration		.09	2.15	.03*
Violence		-.04	-.97	.33	Violence		.03	.70	.50
Model 3	.01				Model 3	.01			
Hassle C E		-.05	-1.38	.17	Hassle C E		.04	.80	.42
Hassle B		.07	1.72	.09	Hassle B		-.06	-1.76	.24
Violence		-.04	-.99	.32	Sleep Duration		.09	2.17	0.30*
Model 4	.01				Model 4	.01			
Hassle C E		-.05	1.56	.12	Hassle B		-.04	-.91	.36
Hassle B		.06	1.65	.10	Sleep Duration		.09	2.21	.03*
Model 5	.002				Model 5	.01			
Hassle B		.06	1.24	.22	Sleep Duration		.09	2.18	.03*

Note: β = standardized beta coefficient, * = significant at p <.05. Hassle LC = Local Community; Hassle CE = Criminal Encounters; and Hassle B = Bullying. Life Stress is not included since there were no measurements done on T2.

As seen in Table 8, the paired sample t-test, there was significant changes among all the stress variables from T1 (2006) to T2 (2008) and the above analysis, table 9, explores if any changes in stress are related to those that become overweight in T3. The results found a significant relationship between boys sleep length and their BMI on T3, while controlling for the other stress variables. In other words, changes in sleep length from more to less sleep on weekdays, significantly predicted increased BMI on T3 among boys.

Discussion

This study has been an attempt to explore the influence of different types of stress factors on weight gain among adolescents in Oslo, in a longitudinal design. The study revealed that the girls that had experienced higher levels of Life Stress initially; were significantly more likely to become overweight three years later. While it may seem plausible that girls who are already overweight in T1 subsequently experience more Life Stress, this development was also found to affect girls that were normal weight in T1, implying that Life Stress predicts higher BMI in girls. Additionally, the study discovered that boys who receive less sleep on weekdays are significantly predicted to be overweight in T3, compared to those that receive more sleep. All stress variables, except Life Stress, which was only measured on T1, had a considerably mean increase between T1 and T2 among both gender. The changes in stress levels were also examined, and reduced sleep in boys, which may indicate higher stress level, were reliably related to overweight in T3. These findings will be further discussed below.

Instantaneously most striking when looking at the results, are the gender differences, girls respond to Life Stress while boys respond to Sleep Duration. What does previous presented data tell us about possible gender differences in the results?

Undertaking the analysis of this study, several gender differences have been observed. There were considerably more boys present in the overweight and obese categories than girls, in T2 and T3. Within the weight categories, crosstabulation also revealed noteworthy weight changes between the genders from 2006 (T1) to 2009 (T3). Normal weight boys in T1 were found to have a higher likelihood of developing overweight and obese status in T3, than girls, and the boys that were already considered overweight in T1, had an increased tendency of staying overweight or becoming obese in T3, compared to girls. These sex differences are contradictory to the findings from the (Hovengen, 2011), who found a larger percentage of girls

overweight, compared to boys, although their results pertained to students in third grade, and thus; they are not truly comparable. On the other hand, these gender differences are congruent with the Young-HUNT study on Norwegian adolescents (Bjornelv, Lydersen, Holmen, Lund Nilsen, & Holmen, 2009).

Overall, gender differences in studies on the subject matter are not consistent among children or adults. Some have found stress to lead to overweight mostly in women, and others have found stress to lead to overweight mostly in men (Korkeila, Kaprio, Rissanen, Koshenvuo, & Sorensen, 1998; M.L. Sulkowski et al., 2011). Thus, there exist gender differences; yet they do not appear to be systematic. This is a possible contribution to the fact that Life Stress predicted overweight in only girls and not in boys; however, it does not explain why only boys seems to react with an elevated BMI on changes in sleep length.

Socio-economic status is another explanatory variable that could contribute to the gender variances. As displayed in table 2, socio-demographic variables, there were a relatively equal dispersion between girls and boys in social class and also in the location of upbringing. Although analysis of variance (ANOVA), found that girls from a section of the Outer East Side weighed significantly more than girls from the Inner West Side. Furthermore, a relationship was found between boys in lower social class in T1 and a higher BMI three years later. Taken together, socio-economic status appears to be a possible explanation for the variances observed. This is not in line with Wang (2001) who found no consensus of this in his review.

Time, could plausibly be a third variable and responsible for the inconsistent gender results found in this phase. Puberty and maturation occurs at different time periods during the teenage phase. This particular study occurs over a 3 years' timeframe and could possibly exclude important maturation factors in some people. As mentioned, the BMI does not differentiate between muscles or adipose tissue, and hence, it is a possibility that an increased BMI is due to muscle development, particularly in boys.

There was a strong medium correlation between Violence and Life Stress. This relation could conceivably be due to an overlap between some of the questions from these two different scales. These variables also covariated in the regression analyses, more specific, Life Stress became significant when Violence was removed. It implies that it is not the Violence part of the Life Stress questions that contributes to higher body weight in girls, but rather other types of

questions in regards to family and social relations etc (see appendix A for more details on these types of questions). Also, in the correlation, Sleep Duration and Life Stress correlated weakly in girls but a stronger correlation was seen in boys. This might explain why Life Stress does not affect boys' BMI, and also that Sleep Duration might be closer connected to stress for boys than for girls. It is difficult to determine exactly what this means, however, it suggests that Sleep Duration might be an indirect measure of stress, but more so with boys, than with girls. There is probably several possible explanations to this, for example that boys might react to stress differently than girls. This should be investigated further.

The above mentioned variables might indeed contribute to the gender inconsistencies found in this study, another possible explanation however could be that there actually exists specific gender differences, in the encounter of stress. This will be explored further, after taking a closer look upon Sleep Duration as a measure of stress, or not, and its association with BMI in boys.

Sleep length and sleep problems might be seen as indirect measures of stress (Patel & Hu, 2008), and the results therefore indicate that stress, measured as Sleep Duration, leads to overweight. There are however, several different explanations that need to be taken into account on this matter. As seen earlier, short sleep length in itself, might lead to overweight or obesity across all age groups. Besides, the relationship between short sleep duration and obesity seems to be even more robust in children (Taheri, 2006), than adults. However, it is difficult to determine whether sleep is an expression of stress, or if merely the length of sleep affects BMI.

A natural inclination towards the gender difference is that it could be explained by WHO's (2011a) causes of obesity, mainly; changes in behavioral and environmental factors that may decrease energy expenditure and increases energy intake. For instance, studies have found gender differences in computer use in secondary school, boys at this age tend to use computers more frequently than girls, and have more positive attitudes towards computers and computer games (Colley & Comber, 2003; Shashaani, 1993). In addition, the ownership of a computer, the usage of it, higher frequency of playing video games, and being on the internet is in fact; associated with children going to bed significantly later and spending significantly less time in bed on weekdays (Van den Bulck, 2004) Consequently, less time sleeping, allows for more time eating, and sedentary activities burns less calories, which may again affect boys' body weight.

Thus, distal causes of obesity could be a possible explanation for the relationship between sleep and BMI among boys. This is in line with Patel and Hu's (2008) causal pathway theory linking sleep with weight gain. However, girls and boys reported approximately the same amount of sleep on weekdays, indicating that girls got as little, if not less sleep, as boys. Nevertheless, girls might be more inclined to care about their looks and might therefore to a greater extent restrain their eating during late hours. Or they may be preoccupied with different types of activities than boys, and these activities may not involve caloric consumption to the same extent. In addition, the activity of playing video or computer games might be seen as a stressful factor in itself. In fact, video games have often been used as a stressor in studies to measure cardiovascular reactivity, and even though this is a disputed subject, it has been found that stressful music in video games may increase cortisol release in adolescents (Hebert, Beland, Dionne-Fournelle, Crete, & Lupien, 2005). Again, this might also explain why Sleep Duration and Life Stress are stronger correlated in boys than in girls.

During the last few years, it is well documented that girls tend to do better in most school courses than boys, as reflected by grades, and this is also true in Norway (Nordahl, Løken, Knudsmoen, Aasen, & Sunnvåg, 2011). This difference in grades between girls and boys tends to cease in high school (Markuss, Frøseth, Lødding, & Sandberg, 2008). Perhaps boys are starting to realize the importance of good grades, and that they have to start working harder to bring their grade point level up. Even though homework is often found to be perceived as more stressful and time consuming to girls (Kouzma & Kennedy, 2002; Owen-Yeates, 2005), girls might do a better job in managing their workspace, and budget their time because of years of experience (Xu, 2006). Thus, girls might have developed techniques to comprehend schoolwork, while boys might tend to procrastinate more, and do their homework in the last minute. It is, for instance, found that while women seem to react more strongly to interpersonal stress rejections, men tend to react more strongly to achievement challenges (Stroud, Salovey, & Epel, 2002). Hence, both girls and boys reported a decrease in sleep length during the three years, and one explanation for this might be more homework. Perhaps a bit speculative, but it might be possible that boys have a more stressful reaction to the achievement challenges they face in high school. However, on the contrary, it might also not affect their psychological wellbeing notably.

The girls in our study showed a higher BMI when exposed to the variable Life Stress, which, compared to Sleep Duration, is a more direct measure of stress. It is interesting however, to see that this exposure predicts higher BMI in girls but not in boys. This could be explained by the notion that girls may be more sensitive to the exposure of Life Stress than boys. Maybe boys and girls experienced the same amount of these stressful life events, except the girls perceived them as more stressful. This notion is consistent with Matud's finding (2004), women scored significantly higher on stress than men, although there was no difference in the number of life events experienced in the previous two years. Also, the women rated their life events as more negative and less controllable then the men. In line with this, it is conceivable that girls and boys hold different coping styles when dealing with the stress of life events. Research has found women to be more likely to use avoidance and emotional coping styles in the face of stress (Billings & Moos, 1981; Matud, 2004; Ptacek, Smith, & Dodge, 1994). Interestingly, as mentioned earlier, Sulkowski and colleagues (2011) found that the coping style one uses when experiencing stress, affects eating habits too - emotion-focused and avoidant coping style were positively associated with stress and binge eating.Which is also in line with behavioral changes and distal causes of obesity (WHO, 2011a) and activation of the stress-system concerned with regulating food intake (Valassi et al., 2008). Not the least, it is congruent with the finding that girls in our study, went from normal weight to overweight, when experiencing Life Stress three years earlier. As previously mentioned, when encountering stress, restrained eaters typically report eating more than non-restrained eaters (Oliver & Wardle, 1999). Furthermore, several studies have found restrained eaters predominantly to be women and not men (Forster & Jeffery, 1986; Klem, Klesges, Bene, & Mellon, 1990; Rand & Kuldau, 1991). This may also explain why girls and not boys in our study develop overweight when facing stressful life events. The inability to maintain control of self-imposed rules concerning food intake when stressed might make these girls eat more than usual and hence gain weight. Oliver and Wardle (1999) also found this in their study, but they found no gender difference in the overall food intake when stressed, hence this subject is disputed and needs to be investigated further.

If girls tend to react more strongly to stressful stimuli, then why are there no connections between Violence, Sleep Duration, Hassles and body weight? Since the Stressful Life Events Scale is developed with the purpose of measuring stress, it makes sense that this measure

predicts changes that the indirect measures of stress Violence and Sleep Duration does not. However, is the measure of Life Stress a more reliable measure of stress than the Urban Hassle Index too? Actually, previous research on these two scales suggests the opposite, that is; Kanner et al. (1981), found that the Hassles Scale was a better predictor of concurrent and subsequent psychological symptoms than were the life events scores, and that the scale shared most of the variance in symptoms accounted for by life events. When the effects of life events scores were removed, hassles and symptoms remained significantly correlated. Nevertheless, there is a chance that this age group does not encounter Daily Hassles in the same way adults does, the experience of stressful life events might be perceived as bigger stressors than the other stress variables. This might also be the explanation for why Hassles and Violence was not associated with higher body weight in boys.

Study Limitations

In spite of all the strengths with the current study, there were several limitations that should be considered when interpreting the results.

First, self-reported information may be inaccurate and thus affect the validity of the study. In particular, under or overestimation on height and weight (BMI) may possibly have imprecise answer. However, graphical representations on the BMI dispersion were conducted early on, and potential outliers were removed from analysis, which reduces the likelihood of incorrect answers. In addition, self- reported information on the variables capturing stress and sleep may also be inaccurate. Social desirability is often found in self-reporting surveys and it is possible that it has tainted this study.

Second, while the BMI is a common measure utilized in weight related research, it can reflect elevations in other aspects of body composition, such as muscle mass, rather than adipose tissue. Natural puberty changes allow boys to develop more muscle mass than girls, and it may not be proportionate to the growth in height. Yet, the tool has been tested out on multiple and large populations, it does separate BMI based on genders, and it is age specific.

Third, subject attrition is a prominent problem in longitudinal designs. Moreover, subject attrition due to drop-out creates a discrepancy between those who remain and those who leave the study. It is possible that the withdrawing participants contain important features that would change the final results in this study. Several steps were done to evaluate discrepancies in

representativeness between the test points. The results revealed that there was a significant gender difference, such that; more boys went missing between the test points. In other terms, more girls participated in the study as whole, which increases the chances of a significant finding. Furthermore, independent t-tests revealed a considerably difference in the average scores for Violence exposure and Life Stress in boys who dropped out of the study after T1. The same tendency, although not significant were found among girls. This selection drop-out bias was controlled by conducting analysis split on gender. However, it is likely that we may have underestimated the effect between Violence exposure and Life Stress on BMI, since those that are exposed to higher level of Violence and Life Stress, left the study after the first test.

Fourth, stress measured in various ways has been found to alter both energy intake and also the development of overweight in all age groups. We have focused on certain different stress measurements or indications; both major stressful life events and hassles have been shown to affect health outcomes in general, and would likely lead to overweight. Even though studies show that the scales that were utilized here - Violence exposure, Sleep Duration and particularly Daily Hassles indeed are measures of stress; there is a chance that our population did not experience them in this way. Rather, in this age group, the experience of major stressful life events might be perceived as superior stressors compared to the other stress variables.

Fifth, the timeframe of the study, three years from 2006 to 2009 might not capture the important changes that we are looking for. Even though, as seen, there is an overall increase in overweight amongst children and youth in Norway, the three years we have studied might not be sufficient to apprehend these transformations.

Sixth, in the LUNO study, 2416 students participated in T1, which constitutes close to 60 percent of the total population asked. In general, the larger the sample size and the higher response rate indicate better estimates, and consequently, less room for a random chance in the results. The study response rates are not excellent and hence; the study may contain selection bias. The target population did not include a large part of its desired participants, roughly 40 percent is missing, and thus it is possibly that the current study has a less representative population, and that the results would differ if they were included.

Finally, as is the case with all longitudinal research, it is likely that some variable unaddressed in this study could explain the prospective effects.

Future Research

It is important to conduct more research on stress in relation to weight gain. For instance, longitudinal studies that cover longer time periods may shed some lights as to onset, maturation, and time in itself. In regards to sleep, it would be beneficial to include studies which examine sleep, individual differences related to self-regulation that revolves around restrained versus non-restrained eaters, coping style and reactivity to stress, energy intake, and physical activity. These types of studies may help detangle the sleep and weight gain relationship with BMI. Additionally, experimental studies that manipulates sleep duration, may clarify if there is a causal relationship between sleep deprivations and overweight. These studies could also rule out the existence of a potential third variable affecting the result.

In conclusion, what is learned from this study is at the least that stress in the form of increased Life Stress affects adolescent girls 'weight over time and shorter Sleep Duration affects adolescent boys' weight over time. There also appears to be multiple gender differences involved. It is evident however, that stress and weight gain has many aspects, and hence, it is difficult to pinpoint exactly which factors are responsible for weight gain among teens.

References

Akerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L., & Kecklund, G. (2002). Sleep disturbances, work stress and work hours: a cross-sectional study. *Journal of Psychosomatic Research, 53*, 741-748.

American Psychological Association. (2011). American Psychological Association: Glossary of Psychological Terms Retrieved October 21, 2011, from http://www.apa.org/research/action/glossary.aspx

Baird, J., Fisher, D., Lucas, P., Kleijnen, J., Roberts, H., & Law, C. (2005). Being big or growing fast: systematic review of size and growth in infancy and later obesity. *British Medical Journal, 331*, 929. doi: 10.1136/bmj.38586.411273.E0

Banks, S., & Dinges, D. F. (2007). Behavioral and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine, 3*, 519-528.

Barlow, J., & Underdown, A. (2005). Promoting the social and emotional health of children: where to now? *Journal of the Royal Society of Health, 125*, 64-70.

Baum, A. (1990). Stress, intrusive imagery, and chronic distress. *Health Psychol, 9*(6), 653-675.

Billings, A. G., & Moos, R. H. (1981). The role of coping responses and social resources in attenuating the stress of life events. *Journal of Behavioral Medicine, 4*, 139-157. doi: 10.1007/bf00844267

Bjorge, T., Engeland, A., Tverdal, A., & Smith, G. D. (2008). Body mass index in adolescence in relation to cause-specific mortality: a follow-up of 230,000 Norwegian adolescents. *American Journal of Epidemiology, 168*, 30-37. doi: 10.1093/aje/kwn096

Bjornelv, S., Lydersen, S., Holmen, J., Lund Nilsen, T. I., & Holmen, T. L. (2009). Sex differences in time trends for overweight and obesity in adolescents: The Young-HUNT study. *Scandinavian Journal of Public Health, 37*, 881-889. doi: 10.1177/1403494809347022

Björntorp, P. (2001). Do stress reactions cause abdominal obesity and comorbidities? *Obesity Review, 2*, 73-86.

Butte, N. F., Garza, C., & de Onis, M. (2007). Evaluation of the feasibility of international growth standards for school-aged children and adolescents. *Journal of Nutrition, 137*, 153-157.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear and rage: an account of recent research into the function of emotional excitement.* (2nd ed.). New York: Appleton-Century-Crofts.

Cappuccio, F. P., Taggart, F. M., Kandala, N. B., Currie, A., Peile, E., Stranges, S., & Miller, M. A. (2008). Meta-analysis of short sleep duration and obesity in children and adults. *Sleep Journal, 31*, 619-626.

Carlson, L. E., & Garland, S. N. (2005). Impact of mindfulness-based stress reduction (MBSR) on sleep, mood, stress and fatigue symptoms in cancer outpatients. *International Journal of Behavioral Medicine, 12*, 278-285. doi: 10.1207/s15327558ijbm1204_9

Carter, P. J., Taylor, B. J., Williams, S. M., & Taylor, R. W. (2011). Longitudinal analysis of sleep in relation to BMI and body fat in children: the FLAME study. *British Medical Journal, 342*. doi: 10.1136/bmj.d2712

Cartwright, M., Wardle, J., Steggles, N., Simon, A. E., Croker, E., & Jarvis, M. J. (2003a). Stress and Dietary Practices in Adolescents. *Journal of Health Psychology, 22*, 362-269. doi: 10.1037/0278-6133.22.4.362

Center for Disease Control. (2012, March 9th - 2012). Healthy weight Retrieved March 09 - 2012, CDC, from http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html#normal

Chan, R. S. M., & Woo, J.,. (2010). Prevention of overweight and obesity: How effective is the current public health approach. *International Journal of Environmental Research and Public Health 7*, 765-783. Retrieved from http://cirrie.buffalo.edu/database/?au=Woo%20J

Charmandari, E., Kino, T., Souvatzoglou, E., & Chrousos, G. P. (2003). Pediatric stress: hormonal mediators and human development. *Journal Hormone Research in Paediatrics, 59*, 161-179.

Charmandari, E., Tsigos, C., & Chrousos, G. (2005). Endocrinology of the stress response. *Annual Review of Physiology, 67*, 259-284.

Chrousos, G. P. (2000). The role of stress and the hypothalamic-pituitary-adrenal axis in the pathogenesis of the metabolic syndrome: neuro-endocrine and target tissue-related causes. *International journal of obesity and related metabolic disorders 24* S50-55.

Clench-Aas, J., Rognerud, M., & Dalgard, O. S. (2009). Levekårsundersøkelsen 2005. Psykisk helse i Norge (D. o. m. health, Trans.). Oslo: The Norwegian National Health Institute.

Colley, A., & Comber, C. (2003). Age and gender differences in computer use and attitudes among secondary school students: what has changed? *Journal of Educational Research, 45*, 155-165. doi: 10.1080/0013188032000103235

Conway, T. L., Vickers, R. R., Jr., Ward, H. W., & Rahe, R. H. (1981). Occupational stress and variation in cigarette, coffee, and alcohol consumption. *Journal of Health and Social Behavior, 22*, 155-165.

Coyne, J., Kanner, A., and Hulley, L. . (1979). *Life events, hassles, and adaptation: A test of the shoelace hypothesis.* Paper presented at the Western Psychological Association, San Diego, California, April 6, 1979.

Crowther, J. H., Snaftner, J., Bonifazi, D. Z., & Shepherd, K. L. (2001). The role of daily hassles in binge eating. *The International Journal of Eating Disorders, 29*, 449-454. doi: 10.1002/eat.1041

Dallman, M. F., Pecoraro, N. C., & la Fleur, S. E. (2005). Chronic stress and comfort foods: self-medication and abdominal obesity. *Brain, Behavior and Immunity, 19*, 275-280. doi: 10.1016/j.bbi.2004.11.004.

Danielsen, Y. S., Pallesen, S., Stormark, K. M., Nordhus, I. H., & Bjorvatn, B. (2010). The relationship between school day sleep duration and body mass index in Norwegian children (aged 10-12). *International Journal of Pediatric Obesity, 5*, 214-220. doi: 10.3109/17477160903473739

De Vriendt, T., Moreno, L. A., & De Henauw, S. (2009). Chronic stress and obesity in adolescents: Scientific evidence and methodological issues for epidemiological research. *Nutrition, Metabolism and Cardiovascular Diseases, 19*, 511-519. doi: 10.1016/j.numecd.2009.02.009.

Dockray, S., Susman, E. J., & Dorn, L. D. (2009). Depression, Cortisol Reactivity, and Obesity in Childhood and Adolescence. *The Journal of adolescent health : official publication of the Society for Adolescent Medicine, 45*, 344-350.

Epel, E., Lapidus, R., McEwen, B., & Brownell, K. (2001). Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. *The Journal of Psychoneuroendocrinology, 26*, 37-49.

Ewart, C. K., & Suchday, S. (2002). Discovering how urban poverty and violence affect health: development and validation of a Neighborhood Stress Index. *Journal of Health Psychology, 21*, 254-262.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., . . . Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. [Research Support, U.S. Gov't, P.H.S.]. *American Journal of Preventative Medicine, 14*, 245-258.

Flegal, K. M., & Ogden, C. L. (2011). Childhood obesity: are we all speaking the same language? *Advances of Nutrition, 2*, 159S-166S. doi: 10.3945/an.111.000307

Forster, J. L., & Jeffery, R. W. (1986). Gender differences related to weight history, eating patterns, efficacy expectations, self-esteem, and weight loss among participants in a weight reduction program. *Addictive Behaviors, 11*, 141-147.

Freeman, L. M. Y., & Gil, K. M. (2004). Daily Stress, coping and dietary restraint in binge eating. . *The International Journal of Eating Disorders, 36*, 204-212. doi: 10.1002/eat.20012

Glozier, N., Martiniuk, A., Patton, G., Ivers, R., Li, Q., Hickie, I., . . . Stevenson, M. (2010). Short sleep duration in prevalent and persistent psychological distress in young adults: the DRIVE study. *Sleep Journal, 33*, 1139-1145.

Goodman, E., & Whitaker, R. C. (2002). A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics, 110*, 497-504.

Goodyer, I. M. (2001). *The depressed child and adolescent, 2nd edn.* Cambridge, UK: Cambridge University Press.

Guh, D. P., Zhang, W., Bansback, N., Amarsi, Z., Birmingham, C. L., & Anis, A. H. (2009). The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. *BioMed Central Public Health, 9*, 88. doi: 10.1186/1471-2458-9-88

Gunnar, M. R., & Cheatham, C. L. (2003). Brain and behavior interface: Stress and the developing brain. *Infant Mental Health Journal, 24*, 195-211. doi: 10.1002/imhj.10052

Haines, J., Neumark-Sztainer, D., Wall, M., & Story, M. (2007). Personal, Behavioral, and Environmental Risk and Protective Factors for Adolescent Overweight. *Obesity, 15*, 2748-2760.

Haugland, S., Wold, B., & Torsheim, T. (2003). Relieving the pressure? The role of physical activity in the relationship between school-related stress and adolescent health complaints. *Research Quarterly for Exercise & Sport, 74*, 127-135.

Heatherton, T. F., Herman, C. P., & Polivy, J. (1991). Effects of physical threat and ego threat on eating behavior. *Journal of Personality and Social Psychology, 60*, 138-143.

Hebert, S., Beland, R., Dionne-Fournelle, O., Crete, M., & Lupien, S. J. (2005). Physiological stress response to video-game playing: the contribution of built-in music. *Life Science, 76*, 2371-2380. doi: 10.1016/j.lfs.2004.11.011

Hinkle, L. E. (1974). The concept of "stress" in the biological and social sciences. *International Journal of Psychiatric Medicine, 5*, 355-357.

Houbre, B., Tarquinio, C., Thuillier, I., & Hergott, E. (2006). Bullying among students and its consequences on health. *European Journal of Psychology of Education, 21*, 183-208. doi: 10.1007/bf03173576

Hovengen, R. S., H. B. (2011). Barns vekst i Norge - resultater 2010 (pp. 1-6). Oslo: Norwegian National Health Institutes.

Kanner, A., & Coyne, J. (1979). *Uplifts, hassles, and a little androgyny.* Paper presented at the Paper presented at the Meeting of the Western Psychological Association, San Diego, California, April 6, 1979.

Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. S. (1981a). Comparison of two modes of stress measurement: daily hassles and uplifts versus major life events. *J Behav Med, 4*, 1-39.

Kanner, A. D., Coyne, J. C., Schaefer, C., & Lazarus, R. S. (1981b). Comparison of two modes of stress measurement: daily hassles and uplifts versus major life events. *Journal of Behavior Medicine, 4*, 1-39.

Kivimaki, M., Head, J., Ferrie, J. E., Shipley, M. J., Brunner, E., Vahtera, J., & Marmot, M. G. (2006). Work stress, weight gain and weight loss: evidence for bidirectional effects of job strain on body mass index in the Whitehall II study. *International Journal of Obesity, 30*, 982-987.

Klem, M. L., Klesges, R. C., Bene, C. R., & Mellon, M. W. (1990). A psychometric study of restraint: The impact of race, gender, weight and marital status. *Addictive Behaviors, 15*, 147-152. doi: 10.1016/0306-4603(90)90018-s

Korkeila, M., Kaprio, J., Rissanen, A., Koshenvuo, M., & Sorensen, T. I. (1998). Predictors of major weight gain in adult Finns: stress, life satisfaction and personality traits. *International Journal of Obesity and Relatated Metabolic Disordorders, 22*, 949-957.

Kouvonen, A., Kivimaki, M., Cox, S. J., Cox, T., & Vahtera, J. (2005). Relationship between work stress and body mass index among 45,810 female and male employees. *Psychosomatic Medicine, 67*, 39-49.

Kouzma, N. M., & Kennedy, G. A. (2002). Homework, Stress, and Mood Disturbance in Senior High School Students. *Psychological Reports, 91*, 193-198. doi: 10.2466/pr0.2002.91.1.193

Kuczmarski, R. J., Ogden, C. L., Guo, S. S., Grummer-Strawn, L. M., Flegal, K. M., Mei, Z., . . . Johnson, C. L. (2002). 2000 CDC Growth Charts for the United States: methods and development. *Vital and Health Statistics Journal*, 1-190.

Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: a review. *Biological Psychology, 69*, 113-132. doi: 10.1016/j.biopsycho.2004.11.009

Laitinen, J., Ek, E., & Sovio, U. (2002). Stress-related eating and drinking behavior and body mass index and predictors of this behavior. *Preventative Medicine, 34*, 29-39. doi: 10.1006/pmed.2001.0948

Lasarus R. S., Folkman S., & (1984). *Stress, Appraisal and Coping*. New York
Springer Pub. Co.

Lazarus, R. S. (1980). *The stress and coping paradigm*. New York: Spectrum.

Lazarus, R. S., Kanner, A., & Folkman, S. (1980). *Emotions: A cognitive-phenomenological analysis*. New York: Academic Press.

Lederbogen, F., Kirsch, P., Haddad, L., Streit, F., Tost, H., Schuch, P., . . . Meyer-Lindenberg, A. (2011). City living and urban upbringing affect neural social stress processing in humans. [10.1038/nature10190]. *Nature, 474*, 498-501. doi: http://www.nature.com/nature/journal/v474/n7352/abs/nature10190-f1.2.html#supplementary-information

Lewis, C. E., McTigue, K. M., Burke, L. E., Poirier, P., Eckel, R. H., Howard, B. V., . . . Pi-Sunyer, F. X. (2009). Mortality, Health Outcomes, and Body Mass Index in the Overweight Range. *Circulation, 119*, 3263-3271. doi: 10.1161/circulationaha.109.192574

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. [10.1038/nrn2639]. *Nature Reviews Neuroscience, 10*, 434-445.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W. J. H., & Zitman, F. G. (2010). Overweight, Obesity, and Depression: A Systematic Review and Meta-analysis of Longitudinal Studies. *Archieves og General Psychiatry, 67*, 220-229. doi: 10.1001/archgenpsychiatry.2010.2

Maher, M. J., Rego, S. A., & Asnis, G. M. (2006). Sleep disturbances in patients with post-traumatic stress disorder: epidemiology, impact and approaches to management. *CNS Drugs, 20*, 567-590.

Markuss, E., Frøseth, M. W., Lødding, B., & Sandberg, N. (2008). Bortvalg og kompetanse. In f. o. u. © NIFU STEP Norsk institutt for studier av innovasjon (Ed.), (Vol. 13/ 2008). Oslo: NOVA.

Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences, 37*, 1401-1415. doi: 10.1016/j.paid.2004.01.010

McCann, B. S., Warnick, G. R., & Knopp, R. H. (1990). Changes in plasma lipids and dietary intake accompanying shifts if perceived workload and stress. *Psychosomatic Medicine, 52*, 97-108.

Meaney, M. J., Diorio, J., Francis, D., Widdowson, J., LaPlante, P., Caldji, C., . . . Plotsky, P. M. (1996). Early Environmental Regulation of Forebrain Glucocorticoid Receptor Gene

Expression: Implications for Adrenocortical Responses to Stress; pp. 61–72. *Developmental Neuroscience, 18*, 61-72.

Mehlum, L. (1999). Alcohol and stress in Norwegian United Nations peacekeepers. *Military Medicine, 164*, 720-724.

Miller, D., & Townsend, A. (2005). Urban Hassles as Chronic Stressors and Adolescent Mental Health: The Urban Hassles Index. *Brief Treatment and Crisis Intervention, 5*, 85-94. doi: 10.1093/brief-treatment/mhi004

Miller, T. Q., Smith, T. W., Turner, C. W., Guijarro, M. L., & Hallet, A. J. (1996b). A meta-analytic review of research on hostility and physical health. *Psychological Bulletin, 119*, 322-348.

Morin, C. M., Rodrigue, S., & Ivers, H. (2003). Role of stress, arousal, and coping skills in primary insomnia. *Psychosomatic Medicine, 65*, 259-267.

Muennig, P., & Bench, K. K. (2009). Obesity-associated stigma and physiological markers of stress: evidence from the Dominican Republic. *Stress and Health, 25*, 241-246. doi: 10.1002/smi.1243

Must, A., Jacques, P. F., Dallal, G. E., Bajema, C. J., & Dietz, W. H. (1992). Long-Term Morbidity and Mortality of Overweight Adolescents. *New England Journal of Medicine, 327*, 1350-1355. doi: doi:10.1056/NEJM199211053271904

Müller-Riemenschneider, F., Reinhold, T., Berghöfer, A., & Willich, S. (2008). Health-economic burden of obesity in Europe. *European Journal of Epidemiology, 23*, 499-509. doi: 10.1007/s10654-008-9239-1

Ng, D. M., & Jeffery, R. W. (2003). Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychology, 22*, 638-642.

Nordahl, T., Løken, G. L., Knudsmoen, H., Aasen, A. M., & Sunnvåg, A. K. (2011). Schools with small gender differences (Vol. 14, pp. 97). Elverum: Hedemark University College.

Norris, R., Carroll, D., & Cochrane, R. (1992). The effects of physical activity and exercise training on psychological stress and well-being in an adolescent population. *Journal of Psychosomatic Research, 36*, 55-65. doi: 10.1016/0022-3999(92)90114-h

Oliver, G., & Wardle, J. (1999). Perceived Effects of Stress on Food Choice. *Physiology & Behavior, 66*, 511-515. doi: 10.1016/s0031-9384(98)00322-9

Owen-Yeates, A. (2005). Stress in Year 11 Students. *Pastoral Care in Education, 23*, 42-51. doi: 10.1111/j.1468-0122.2005.00350.x

Ozer, E. J., & Weinstein, R. S. (2004). Urban Adolescents' Exposure to Community Violence: The Role of Support, School Safety, and Social Constraints in a School-Based Sample of Boys and Girls. *Journal of Clinical Child & Adolescent Psychology, 33*, 463-476. doi: 10.1207/s15374424jccp3303_4

Parsons, T. J., Power, C., Logan, S., & Summerbell, C. D. (1999). Childhood predictors of adult obesity: a systematic review. *International Journal of Obesity Relatated Metabolic Disorders, 23 Suppl 8*, S1-107.

Patel, S. R., & Hu, F. B. (2008). Short sleep duration and weight gain: a systematic review. *Obesity 16*, 643-653. doi: 10.1038/oby.2007.118

Paykel, E. S., Abbott, R., Jenkins, R., Brugha, T. S., & Meltzer, H. (2000). Urban-rural mental health differences in great Britain: findings from the national morbidity survey. *Psychological Medicine, 30*, 269-280.

Polivy, J., Herman, C. P., & McFarlane, T. (1994). Effects of Anxiety on Eating: Does Palatability Moderate Distress-Induced Overeating in Dieters? *Journal of Abnormal Psychology, 103*, 505-510.

Pollard, T. M. (1997). Physiological consequences of everyday psychosocial stress. *Collegium Antropologicum, 21*, 17-28.

Ptacek, J. T., Smith, R. E., & Dodge, K. L. (1994). Gender Differences in Coping with Stress: When Stressor and Appraisals Do Not Differ. *Personality and Social Psychology Bulletin, 20*, 421-430. doi: 10.1177/0146167294204009

Puhl, R. M., & Heuer, C. A. (2009). The stigma of obesity: a review and update. *Obesity 17*, 941-964. doi: 10.1038/oby.2008.636

Puhl, R. M., & Latner, J. D. s. (2007). Stigma, obesity, and the health of the nation's children. *Psychological Bulletin, 133*, 557-580. doi: 10.1037/0033-2909.133.4.557

Rand, C. S. W., & Kuldau, J. M. (1991). Restrained eating (weight concerns) in the general population and among students. *International Journal of Eating Disorders, 10*, 699-708. doi: 10.1002/1098-108x(199111)10:6<699::aid-eat2260100608>3.0.co;2-0

Rehkopf, D. H., Laraia, B. A., Segal, M., Braithwaite, D., & Epel, E. (2011). The relative importance of predictors of body mass index change, overweight and obesity in adolescent girls. *International Journal of Pediatric Obesity, 6*, e233-242. doi: 10.3109/17477166.2010.545410

Roland, E., & Idsøe, T. (2001). Aggression and bullying. *Aggressive Behavior, 27*, 446-462. doi: 10.1002/ab.1029

Schoenborn, C. A., & Adams, P. E. (2010). Health behaviors of adults: United States, 2005-2007. *Vital and Health Statistics Journal*, 1-132.

Schotte, D. E., Cools, J., & McNally, R. J. (1990). Film-Induced Negative Affect Triggers Overeating in Restrained Eaters. *Journal of Abnormal Psychology, 99*, 317-320.

Sebo, P., Beer-Borst, S., Haller, D. M., & Bovier, P. A. (2008). Reliability of doctors' anthropometric measurements to detect obesity. *Preventative Medicine, 47*, 389-393. doi: 10.1016/j.ypmed.2008.06.012

Shashaani, L. (1993). Gender-based differences in attitudes toward computers. *Computers & Education, 20*, 169-181. doi: 10.1016/0360-1315(93)90085-w

Sherwood, L. (2001). *Human physiology from cells to systems.* (4th ed.). California: Brooks/Cole.

Slochower, J., Kaplan, S. P., & Mann, L. (1981). The effects of life stress and weight on mood and eating. *Appetite, 2*, 115-125.

Soldatos, C. R., Allaert, F. A., Ohta, T., & Dikeos, D. G. (2005). How do individuals sleep around the world? Results from a single-day survey in ten countries. *Sleep Medicine, 6*, 5-13. doi: 10.1016/j.sleep.2004.10.006

Sorensen, E., & Ursin, R. (2001). [Sleep habits among adolescents]. *Tidsskrift for den norske Legeforening, 121*, 331-333.

Stice, E., Presnell, K., Shaw, H., & Rohde, P. (2005). Psychological and behavioral risk factors for obesity onset in adolescent girls: a prospective study. *Journal of Consulting and Clinical Psychology, 73*, 195-202. doi: 10.1037/0022-006x.73.2.195

Stone, A. A., & Brownell, K. (1994). The stress-eating paradox: multiple daily measurements in adult males and females. *Psychological Health, 9*, 425-436.

Strand, B. H., Kuh, D., Shah, I., Guralnik, J., & Hardy, R. (2012). Childhood, adolescent and early adult body mass index in relation to adult mortality: results from the British 1946 birth cohort. *Journal of Epidemiology and Community Health, 66*, 225-232. doi: 10.1136/jech.2010.110155

Stroud, L. R., Salovey, P., & Epel, E. S. (2002). Sex differences in stress responses: social rejection versus achievement stress. *Biological Psychiatry, 52*, 318-327. doi: 10.1016/s0006-3223(02)01333-1

Suchday, S., Kapur, S., Ewart, C. K., & Friedberg, J. P. (2006). Urban stress and health in developing countries: development and validation of a neighborhood stress index for India. *Behavior Medicine, 32*, 77-86. doi: 10.3200/bmed.32.3.77-86

Sulkowski, M. L., Dempsey, J., & Dempsey, A. G. (2011). Effects of stress and coping on binge eating in female college students. *Eating Behaviors, ss12*, 188-191. doi: 10.1016/j.eatbeh.2011.04.006

Sumter, S. R., Bokhorst, C. L., Miers, A. C., Van Pelt, J., & Westenberg, P. M. (2010). Age and puberty differences in stress responses during a public speaking task: do adolescents grow more sensitive to social evaluation? *Psychoneuroendocrinology, 35*, 1510-1516. doi: 10.1016/j.psyneuen.2010.05.004

Sund, A. M., Larsson, B., & Wichstrøm, L. (2003). Psychosocial correlates of depressive symptoms among 12–14-year-old Norwegian adolescents. *Journal of Child Psychology and Psychiatry, 44*, 588-597. doi: 10.1111/1469-7610.00147

Sørensen, T. I., Holst, C., & Stunkard, A. J. (1992). Childhood body mass index - genetic and familial environmental influences assessed in a longitudinal adoption study. *International Journal of Obesity, 16*, 705 - 714.

Taheri, S. (2006). The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity. *Archives of Disease in Childhood, 91*, 881-884. doi: 10.1136/adc.2005.093013

Tataranni, P. A., Larson, D., Snitker, S., Young, J., Flatt, J., & Ravussin, E. (1996). Eddects of glucocorticoid on energy metabolism and food intake in humans. *American Journal of Physiology., 271*, E317-E325.

The National Sleep Foundation. (2012). The National Sleep Foundation

How much sleep do we really need? Retrieved 09.02.2012, 2012, from http://www.sleepfoundation.org/article/how-sleep-works/how-much-sleep-do-we-really-need

The, N. S., Suchindran, C., North, K. E., Popkin, B. M., & Gordon-Larsen, P. (2010). Association of Adolescent Obesity With Risk of Severe Obesity in Adulthood. *The Journal of the American Medical Association, 304*, 2042-2047. doi: 10.1001/jama.2010.1635

The World Health Organization. (1996). WHO Global Consultation on violence and health. Violence: a public health priority *WHO Global Consultation on violence and health. Violence: a public health priority* (pp. 372). Geneva: World Health Organization.

The World Health Organization. (2011a). WHO World Health Organization: Global Database on Body Mass Index Retrieved October 1, 2011, from http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

The World Health Organization. (2011b, March 2011s). World Health Organization: Obesity and overweight Retrieved October 1, 2011, from http://www.who.int/mediacentre/factsheets/fs311/en/

Torres, S. J., & Nowson, C. A. (2007). Relationship between stress, eating behavior and obesity. *Nutrition, 23*, 887-894. doi: 10.1016/j.nut.2007.08.008.

Travison, T. G., O'Donnell, A. B., Araujo, A. B., Matsumoto, A. M., & Mckinlay, J. B. (2007). Cortisol levels and measures of body composition in middle aged and older men. *Clinical Endocrinology, 67*, 71-77.

Tsigos, C., & Chrousos, G. P. (2006). Stress, obesity, and the metabolic syndrome - soul and metabolism. *Annals of the New York Academy of Sciences, 1083*, 11-13.

Valassi, E., Scacchi, M., & Cavagnini, F. (2008). Neuroendocrine control of food intake. *Nutrition, Metabolism and Cardiovascular Diseases, 18*, 158-168. doi: 10.1016/j.numecd.2007.06.004.

Van Cauter, E., & Knutson, K. L. (2008). Sleep and the epidemic of obesity in children and adults. *Eur J Endocrinol, 159 Suppl 1*, S59-66. doi: 10.1530/eje-08-0298

Van den Bulck, J. (2004). Television viewing, computer game playing, and Internet use and self-reported time to bed and time out of bed in secondary-school children. *Sleep, 27*, 101-104.

Van Strien, T., Rookus, M. A., Bergers, G. P., Frijters, J. E., & Defares, P. B. (1986). Life events, emotional eating and change in body mass index. *International Journal of Obesity, 10*, 29-35.

Wang, Y. (2001). Cross-national comparison of childhood obesity: the epidemic and the relationship between obesity and socioeconomic status. *International Journal of Epidemiology, 30*, 1129-1136.

Weigensberg, M. J., Toledo-Corral, C. M., & Goran, M. I. (2008). Association between the Metabolic Syndrome and Serum Cortisol in Overweight Latino Youth. *Journal of Clinical Endocrinology & Metabolism, 93*, 1372-1378. doi: 10.1210/jc.2007-2309

Weinstein, S. E., Shide, D. J., & Rolls, B. J. (1997). Changes in food intake in response to stress in men and women: psychological factors. *Appetite, 28*, 7-18. doi: 10.1006/appe.1996.0056

Wilfley, D. E., Vannucci, A., & White, E. K. (2010). Early intervention of eating- and weight-related problems. *Journal of Clinical Psychology in Medical Settings, 17*, 285-300. doi: 10.1007/s10880-010-9209-0

Wilhelmsen, M. (2009). Health Interview Survey 2008/Samordnet levekårsundersøkelse 2008 (pp. 1-138): Statistics Norway.

Willenbring, M. L., Levine, A. S., & Morley, J. E. (1986). Stress induced eating and food preference in humans: A pilot study. *International Journal of Eating Disorders, 5*, 855-864. doi: 10.1002/1098-108x(198607)5:5<855::aid-eat2260050507>3.0.co;2-o

Wittchen, H. U., & Jacobi, F. (2005). Size and burden of mental disorders in Europe - a critical review and appraisal of 27 studies. *European Neuropsychopharmacology, 15*, 357-376. doi: 10.1016/j.euroneuro.2005.04.012.

Wolff, G. E., Crosby, R. D., Roberts, J. A., & Wittrock, D. A. (2000). Differences in daily stress, mood, coping, and eating behavior in binge eating and nonbinge eating college women. *Addictive Behaviors, 25*, 205-216. doi: 10.1016/s0306-4603(99)00049-0

World Health Organization. (2012). World Health Organization: World Health Report 2002. *Health: Obesity*, 2012, from http://www.who.int/topics/obesity/en/

Xu, J. (2006). Gender and Homework Management Reported by High School Students. *Educational Psychology, 26*, 73-91. doi: 10.1080/01443410500341023

Yach, D., Stuckler, D., & Brownell, K. D. (2006). Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. *Nat Med, 12*(1), 62-66. doi: nm0106-62 [pii]10.1038/nm0106-62

Yin, Z., Davis, C. L., Moore, J. B., & Treiber, F. A. (2005). Physical activity buffers the effects of chronic stress on adiposity in youth. *Annual Behavior Medicine, 29*, 29-36. doi: 10.1207/s15324796abm2901_5

Appendices

Appendix 1. The questionnaires which was utilized

Stress - Livshendelser: EASQ (Early Adolescent Stress Questionnaire) oversatt og tilpasset norske 12-14 åringer (Sund, Larsson, & Wichström, 2003)

Har noe av dette noen gang hendt deg? Nedenfor er nevnt livshendelser som ungdom kan ha opplevd i løpet av livet. Sett kryss for ja eller nei ved hver hendelse

	Ja	Nei
Du hadde i en periode mer lekser enn du greide	☐	☐
Læreren gjorde narr av deg foran klassen	☐	☐
Du begynte med spesielt tilrettelagt undervisning	☐	☐
Du fikk ikke nok igjen for innsatsen din på skolen (lite skryt/ for dårlige karakterer)	☐	☐
Du fikk i en periode ikke nok hjelp med skolearbeidet (hjemme eller på skolen)	☐	☐
Du ble ikke tatt ut til laget (skole eller idrettslag) i konkurranser/kamper (håndball, fotball, ski etc.)	☐	☐
Foreldrene dine bekymret seg for mye for skolearbeidet ditt	☐	☐
Du hørte at foreldrene dine kranglet voldsomt eller sloss	☐	☐
Du og foreldrene dine hadde en eller flere voldsomme krangler	☐	☐
Du eller en av dine nærmeste ble utsatt for en kriminell handling, ble frastjålet noe verdifullt, overfalt eller lignende	☐	☐
Du ble tatt for å ha gjort noe galt (stjålet noe eller lignende)	☐	☐
Du ble utsatt for rasisme	☐	☐

Du ble alvorlig syk/alvorlig skadet	☐	☐
Din mor er blitt alvorlig syk/ alvorlig skadet	☐	☐
Din far er blitt alvorlig syk/ alvorlig skadet	☐	☐
En av dine søsken er blitt alvorlig syk/ alvorlig skadet	☐	☐
Dine foreldre ble separerert/skilt	☐	☐

Du måtte velge mellom hvilken av foreldrene dine du ville bo hos	☐	☐
En av foreldrene dine giftet seg på nytt eller fikk samboer	☐	☐
Du fikk søsken/stesøsken	☐	☐

Kjæledyret ditt døde	☐	☐
Du flyttet	☐	☐
Familien hadde alvorlige økonomiske problemer	☐	☐
Din mor eller far mistet jobben (ble arbeidsløs eller permittert)	☐	☐

Du mistet en venn/venninne eller det ble slutt med kjæresten	☐	☐
Du ble utsatt for seksuelt press	☐	☐
En av dine venner var i alvorlig trøbbel	☐	☐
En av dine søsken var i alvorlig trøbbel	☐	☐

Foreldrene dine var mye borte hjemmefra (pga arbeid eller annet)	☐	☐

Du hadde for mye ansvar hjemme (for småsøsken, husarbeid etc.) ☐ ☐

Du var bekymret fordi noen i familien din bruker for mye rusmidler (alkohol, piller, stoff) ☐ ☐

Noen du var glad i døde (slektning, god venn) ☐ ☐

Hvem? …….

Har noe annet hendt deg som har vært vanskelig?

Skriv:

……

…….

Daglig stress: Deler av Urban Hassles Index (D. B. Miller & A. Townsend, 2005) tilpasset norske forhold

Hvor ofte hender følgende med deg der du bor?

Nedenfor er det ramset opp noen erfaringer folk kan ha. Sett ett kryss for hver linje. Tenk tilbake på de 2 siste ukene og kryss av for hvor ofte du har opplevd at du har...

Siste to ukene, hvor ofte har du…	Aldri	Noen gang er	Oft e	Veldi g ofte
…vært engstelig fordi området jeg bor i ikke er trygt	☐	☐	☐	☐
…vært redd for å komme i bråk med ukjente i området der jeg bor	☐	☐	☐	☐
…hørt høylydte og bråkete biler, fester eller naboer om natten	☐	☐	☐	☐
…vært redd for eldre ungdommer eller voksne som står og henger på gatehjørner eller utenfor butikker i området der jeg bor	☐	☐	☐	☐

Hvor ofte har dette hendt deg i løpet av de siste to ukene?

De siste to ukene, hvor ofte har du opplevd…	Aldri	Noen ganger	Ofte	Veldig ofte
At en narkoman har tigget penger av deg	☐	☐	☐	☐
Å måtte ta en omvei til skolen for å unngå bråk	☐	☐	☐	☐
Å bli ertet på grunn av gode karakterer	☐	☐	☐	☐
Å være bekymret for at noen vil stjele klær, penger eller mobil fra deg	☐	☐	☐	☐
Å bli stoppet og tilsnakket av politiet	☐	☐	☐	☐
At ansatte har fulgt etter deg inne i butikken fordi de er mistenksomme	☐	☐	☐	☐
Å ikke kunne gå inn i en matbutikk sammen med venner fordi de har regler som nekter skoleelever adgang eller å være flere enn to stykker	☐	☐	☐	☐
Å bli presset til å slåss av venner	☐	☐	☐	☐
Å bli ertet på grunn av dårlige karakterer	☐	☐	☐	☐
Å ha følt det nødvendig å ha med et våpen (f.eks. en kniv) til beskyttelse	☐	☐	☐	☐
At foreldrene dine har snoket i hva du gjør	☐	☐	☐	☐

Søvn og søvnvaner: spørsmål utviklet ved Kompetansesenteret for studiet av søvnproblemer, som er tilknyttet Universitetet i Bergen.

Når pleier du vanligvis å legge deg på hverdager?	Kl._____ : _____
Når pleier du vanligvis å stå opp på hverdager?	Kl. _____ : _____
Når pleier du vanligvis å legge deg i helger og på fridager ellers?	Kl. _____ : _____
Når pleier du vanligvis å stå opp i helger og på fridager ellers?	Kl. _____ : _____

Hvor mye sover du?

Hvor mye søvn får du vanligvis per døgn på vanlige ukedager?	_____timer
Hvor mye søvn får du vanligvis per døgn i helger og andre fridager/ferier?	_____timer
Hvor mye søvn mener du at du trenger per døgn for å bli ordentlig uthvilt?	_____timer

---

I løpet av den siste måneden, hvor mange dager har du... Sett ett kryss for hver linje	0-5 dager	6-10 dager	11-20 dager	21-25 dager	26-30 dager
…hatt problemer med å sovne etter at lysene ble slukket?	☐	☐	☐	☐	☐
…våknet alt for tidlig uten å få sove igjen?	☐	☐	☐	☐	☐
…følt deg for lite uthvilt etter å ha sovet?	☐	☐	☐	☐	☐
vært så søvnig/trett at det har gått ut over skole eller fritid?	☐	☐	☐	☐	☐
…hatt vansker med å våkne om morgenen når du skulle?	☐	☐	☐	☐	☐
…ikke klart å sovne før kl 0200 (om natten)	☐	☐	☐	☐	☐

Appendix B – The Factor Analysis used to uncover three different Hassle factors.

Factor Analysis

Notes

Output Created		17-Nov-2011 12:01:49
Comments		
Input	Data	M:\pc\Dokumenter\Prosjekt\LUNO\Data\LUNO_T123.sav
	Active Dataset	DataSet1
	Filter	<none>
	Weight	<none>
	Split File	<none>
	N of Rows in Working Data File	2479
Missing Value Handling	Definition of Missing	MISSING=EXCLUDE: User-defined missing values are treated as missing.
	Cases Used	LISTWISE: Statistics are based on cases with no missing values for any variable used.

Syntax	FACTOR /VARIABLES HASSLE1_1 HASSLE1_2 HASSLE1_3 HASSLE1_4 HASSLE1_5 HASSLE1_6 HASSLE1_7 HASSLE1_8 HASSLE1_9 HASSLE1_10 HASSLE1_11 HASSLE1_12 HASSLE1_13 HASSLE1_14 HASSLE1_15 /MISSING LISTWISE /ANALYSIS HASSLE1_1 HASSLE1_2 HASSLE1_3 HASSLE1_4 HASSLE1_5 HASSLE1_6 HASSLE1_7 HASSLE1_8 HASSLE1_9 HASSLE1_10 HASSLE1_11 HASSLE1_12 HASSLE1_13 HASSLE1_14 HASSLE1_15 /PRINT UNIVARIATE EXTRACTION ROTATION /FORMAT SORT BLANK(.10) /PLOT EIGEN /CRITERIA FACTORS(3) ITERATE(25) /EXTRACTION PC /CRITERIA ITERATE(25) /ROTATION VARIMAX /METHOD=CORRELATION.

Resources	Processor Time	00:00:00.390
	Elapsed Time	00:00:00.405
	Maximum Memory Required	28528 (27,859K) bytes

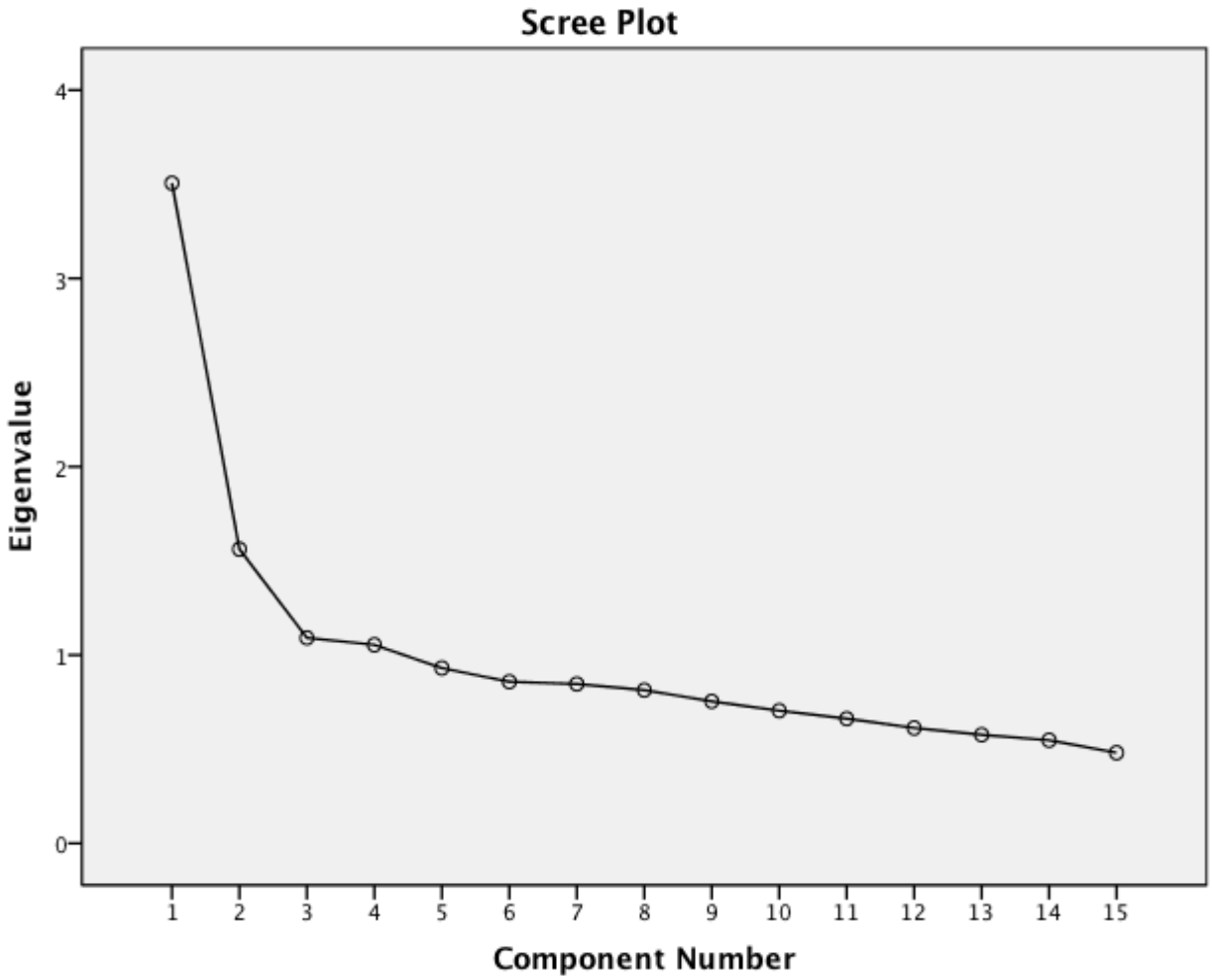
[DataSet1] M:\pc\Dokumenter\Prosjekt\LUNO\Data\LUNO_T123.sav

Descriptive Statistics

	Mean	Std. Deviation	Analysis N
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært engstelig fordi området jeg bor i ikke er trygt?	1.22	.500	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for å komme i bråk med ukjente i området der jeg bor?	1.22	.521	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du hørt høylydte og bråkete biler, fester eller naboer om natten?	1.56	.718	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for eldre ungdommer eller voksne som står og henger på gatehjørner/butikker der du bor?	1.33	.621	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at en narkoman har tigget penger av deg?	1.43	.693	2222

T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å måtte ta en omvei til skolen for å unngå bråk?	1.05	.288	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av gode karakterer?	1.17	.471	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å være bekymret for at noen vil stjele klær, penger eller mobil fra deg?	1.32	.605	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli stoppet og tilsnakket av politiet?	1.08	.339	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at ansatte har fulgt etter deg inne i butikken fordi de er mistenksomme?	1.13	.426	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ikke kunne gå inn i en matbutikk sammen med venner fordi de nekter fler enn to stykker	1.92	1.089	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli presset til å slåss av venner?	1.08	.350	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av dårlige karakterer?	1.11	.383	2222
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ha følt det nødvendig å ha med et våpen (f.eks. en kniv) til beskyttelse?	1.06	.320	2222

T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at foreldrene dine har snoket i hva du gjør?	1.36	.659	2222



Component Matrixa

	Component		
	1	2	3
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for å komme i bråk med ukjente i området der jeg bor?	.619	-.447	-.183
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært engstelig fordi området jeg bor i ikke er trygt?	.607	-.439	-.241
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å måtte ta en omvei til skolen for å unngå bråk?	.540	-.234	.145
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ha følt det nødvendig å ha med et våpen (f.eks. en kniv) til beskyttelse?	.539	.349	-.168
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli presset til å slåss av venner?	.538	.128	.403
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for eldre ungdommer eller voksne som står og henger på gatehjørner/butikker der du bor?	.536	-.505	-.153

T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at ansatte har fulgt etter deg inne i butikken fordi de er mistenksomme?	.504	.427	-.180
T1 Urban Hassle : Siste to uker -, hvor ofte har du hørt høylydte og bråkete biler, fester eller naboer om natten?	.473	-.117	-.259
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å være bekymret for at noen vil stjele klær, penger eller mobil fra deg?	.472		.288
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli stoppet og tilsnakket av politiet?	.471	.457	-.279
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at foreldrene dine har snoket i hva du gjør?	.466	.288	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at en narkoman har tigget penger av deg?	.366	.330	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ikke kunne gå inn i en matbutikk sammen med venner fordi de nekter fler enn to stykker	.271	.310	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av gode karakterer?	.281	-.149	.571
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av dårlige karakterer?	.415		.416

Extraction Method: Principal Component Analysis.

a. 3 components extracted.

Communalities

	Extraction
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært engstelig fordi området jeg bor i ikke er trygt?	.619
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for å komme i bråk med ukjente i området der jeg bor?	.616
T1 Urban Hassle : Siste to uker -, hvor ofte har du hørt høylydte og bråkete biler, fester eller naboer om natten?	.304
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for eldre ungdommer eller voksne som står og henger på gatehjørner/butikker der du bor?	.565
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at en narkoman har tigget penger av deg?	.246
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å måtte ta en omvei til skolen for å unngå bråk?	.367
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av gode karakterer?	.427
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å være bekymret for at noen vil stjele klær, penger eller mobil fra deg?	.307

T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli stoppet og tilsnakket av politiet?	.508
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at ansatte har fulgt etter deg inne i butikken fordi de er mistenksomme?	.469
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ikke kunne gå inn i en matbutikk sammen med venner fordi de nekter fler enn to stykker	.170
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli presset til å slåss av venner?	.468
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av dårlige karakterer?	.351
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ha følt det nødvendig å ha med et våpen (f.eks. en kniv) til beskyttelse?	.440
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at foreldrene dine har snoket i hva du gjør?	.301

Extraction Method: Principal Component Analysis.

Total Variance Explained

Component	Extraction Sums of Squared Loadings			Rotation Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of Variance	Cumulative %
1	3.506	23.373	23.373	2.321	15.471	15.471
2	1.562	10.415	33.789	2.224	14.824	30.295
3	1.091	7.274	41.063	1.615	10.769	41.063

Rotated Component Matrixa

	Component		
	1	2	3
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært engstelig fordi området jeg bor i ikke er trygt?	.775		
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for å komme i bråk med ukjente i området der jeg bor?	.766		.152
T1 Urban Hassle : Siste to uker -, hvor ofte har du vært redd for eldre ungdommer eller voksne som står og henger på gatehjørner/butikker der du bor?	.738		.143
T1 Urban Hassle : Siste to uker -, hvor ofte har du hørt høylydte og bråkete biler, fester eller naboer om natten?	.485	.263	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å måtte ta en omvei til skolen for å unngå bråk?	.449	.107	.393
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli stoppet og tilsnakket av politiet?	.118	.701	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at ansatte har fulgt etter deg inne i butikken fordi de er mistenksomme?	.121	.672	

T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ha følt det nødvendig å ha med et våpen (f.eks. en kniv) til beskyttelse?	.189	.630	
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at foreldrene dine har snoket i hva du gjør?	.128	.505	.173
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd at en narkoman har tigget penger av deg?		.452	.206
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å ikke kunne gå inn i en matbutikk sammen med venner fordi de nekter fler enn to stykker		.393	.121
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av gode karakterer?			.644
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli presset til å slåss av venner?	.112	.311	.599
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å bli ertet på grunn av dårlige karakterer?		.195	.556
T1 Urban Hassle : Siste to uker -, hvor ofte har du opplevd å være bekymret for at noen vil stjele klær, penger eller mobil fra deg?	.225	.173	.476

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization.

a. Rotation converged in 5 iterations.

Component Transformation Matrix

Component	1	2	3
1	.653	.598	.465
2	-.652	.756	-.057
3	-.386	-.266	.883

Extraction Method: Principal Component Analysis.

Rotation Method: Varimax with Kaiser Normalization.